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Clinicopathological Conference
Twelve Year Old Caucasian Male With Asymptomatic
Hypertension

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Table of Contents: page 3

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SCIENTIFIC ARTICLES

- 7 Clinicopathological Conference
Twelve Year Old Caucasian Male With Asymptomatic
Hypertension

M. Stassen, M.D.
J. R. Reynolds, M.D.
J. F. Barlow, M.D.

- 21 Ob-Gyn Lecture #11
Preeclampsia

Loren P. Petersen, M.D.

- 35 Poor Correlation Of Symptoms, Roentgenograms And
Pulmonary Functions In A Case of Endobronchial
Sarcoidosis

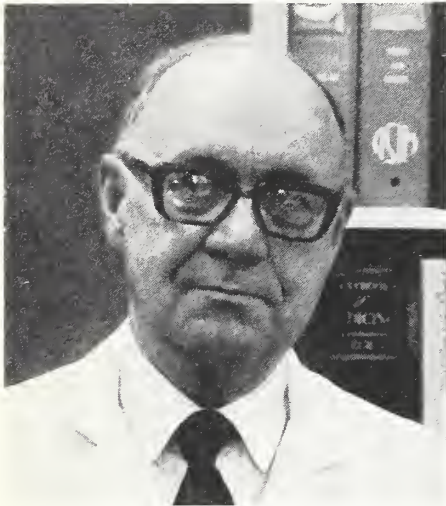
Jay Bachmayer, M.D.
Louis W. Burgher, M.D.

FEATURES

- 18 Laboratory Aids
South Dakota Pathology Page
- 29 South Dakota AFP Chapter News
- 31 This Is Your Medical Association
- 32 Letter To The Editor
- 41 Future Meetings

"THE PHYSICIAN IS A DECISION MAKER, AND ALMOST EVERY DECISION HE MAKES COSTS OR SAVES MONEY."

—Dr. William Felts, Past President,
American Society of Internal Medicine



More and more physicians today are beginning to realize the extent of the economic influence they have, and are finding ways of holding costs down.

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More physicians today realize what a tough problem we're all faced with. They know this is a challenge for medicine. And that physicians are in the best position to deal with and solve the problem.

*PATIENT CARE Magazine—Outlook 1977, "Face-Off: Cost Containment vs. Chaos," January 1, 1977

Lyle CB, et al. "Practice habits in a group of eight internists," ANNALS OF INTERNAL MEDICINE 84 (May 1976), 594-601.

Schroeder SA, et al. "Use of laboratory tests and pharmaceuticals: variation among physicians and effect of cost audit on subsequent use," JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION 225 (Aug. 20, 1973), 969-73



SOUTH DAKOTA BLUE SHIELD
1601 West Madison
Sioux Falls, South Dakota

Twelve Year Old Caucasian Male With Asymptomatic Hypertension

M. Stassen, M.D.*

J. R. Reynolds, M.D.**

Discussers

J. F. Barlow, M.D.***

Editor

Case No. 752334

This 12-year-old Caucasian male was admitted to Sioux Valley Hospital with a chief complaint of hypertension.

The child had a long history of allergic disease with hay fever to substances such as oats, corn, and chocolate and had had repeated attacks of rhinorrhea. This only partly responded to antihistamines. The child had been hospitalized within the month prior to admission because of pneumonia. He had been treated recently with antihistaminics for a swollen eye.

PHYSICAL EXAMINATION: Height 4' 6", weight 85 lbs., pulse 108 per minute, and regular, respirations 20 per minute and regular. Blood pressure 150 systolic and 110 diastolic (admission). The child was well nourished and in no distress. Examination of the head and neck was unremarkable. There was some tortuosity of the retinal vessels on fundic examination. The lungs were clear to auscultation and percussion. The heart showed a Grade III (six grades) systolic ejection murmur heard over the left precordium with transmission into the back and neck. There was an S4 gallop, S1 and S2 were otherwise within normal limits. There were no rubs. The blood pressure was 150 systolic and 100 diastolic in both upper extremities. There were no palpable femoral pulses and a doppler pressure in the lower extremities was 90 systolic. The abdomen revealed no palpable organs, masses, spasm, or tenderness. The testes were descended. Neurologic examination was within normal limits. The patient did not have short stature but may have had webbing of the neck. There was no increased carrying angle of the arms.

LABORATORY DATA: Urinalysis; straw colored, cloudy; specific gravity 1.026; pH 5.0; negative for protein, glucose, reducing substances, ketone bodies, hemoglobin; sediment, negative; hemoglobin 14.9 gms/dl; red blood count 5.58 million/mm³, hematocrit 43 vols/dl, mean corpuscular

hemoglobin 26 micromicrograms, mean corpuscular volume 77 cubic micra, mean corpuscular hemoglobin concentration 35%. White count 5,500/mm³ with 42% segmented neutrophils, 2% neutrophilic bands, 6% eosinophils, 50% lymphocytes. The platelets were normal in number and morphology and the red cells showed slight anisocytosis but were normochromic. pH 7.35, PCO₂ 52 Torr, CO₂ content 28 mm/L. Sodium 135 meq/L, potassium 3.7 meq/L, chloride 100 meq/L, prothrombin time 12 seconds, with a control of 12.5 seconds, partial thromboplastin time 42 seconds with a control of 36 seconds. Lactic dehydrogenase, alkaline phosphatase, aspartate aminotransferase, total bilirubin, calcium, total protein, inorganic phosphorous, glucose, blood urea nitrogen, creatinine, uric acid, cholesterol were within normal limits for age. An electrocardiogram was read as normal for age. An echocardiogram showed very minimal early systolic mitral prolapse. There was no evidence of mitral insufficiency. There was dilatation of the internal dimension of left ventricle to 5 cm. and increased thickness to the septum and left ventricular wall. There was no ptosis. A diagnostic procedure was performed followed by an operation.

DR. STASSEN: I feel that this patient had a classic case of coarctation of the aorta. The presence of this entity gives me a good chance to discuss both hypertension in childhood and coarctation of the aorta.

There is mention in the protocol of webbing of the neck. This suggests Turner's syndrome which has an association with coarctation of the aorta. Pulmonic stenosis and ventricular septal defects occur in Noonan's syndrome which consists of the features of Turner's syndrome in phenotypic males. Patients with Turner's syndrome are usually phenotypic females. These patients have an abnormal chromosome constitution and streak ovaries with infantilism of the genitalia. The karyotype is usually 45 XO although various mosaic patterns such as XO/XX, XX/XY, XO/XYY and XO/XX/XY also occur in phenotypic females.

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Male Turner's syndrome (Noonan's syndrome) usually have 46 chromosomes with a normal karyotype, but XX/XXY and XXXY patterns have been reported.

Systemic hypertension occurs with some frequency in both children and adolescents; and, in a fair population of these patients, it is primary or essential in type. Furthermore, evidence is accumulating which suggests that those patients who are at risk of developing sustained essential hypertension in adult life may be identifiable at an early age.

What is the real definition of pediatric hypertension? At this time it is somewhat unclear. It has been suggested by many authors that if the systolic and diastolic pressures are consistently above the 90th percentile for age that hypertension is thus defined. Loggie, a renowned writer in pediatric hypertension, suggests the following breakdown: 1) If a patient has repeated supine diastolic blood pressures in the 90-95th percentile, these patients are to be defined as borderline hypertension. 2) Definite systemic hypertension is defined as having supine diastolic blood pressures consistently greater than the 95th percentile for age in children and greater than the 90th percentile in adolescence. It must be mentioned that, in Loggie's experience, blood pressures taken sitting or standing are significantly higher than supine diastolic pressures in some individuals. The distribution of blood pressure in various positions in boys and girls has been established.

How prevalent is pediatric hypertension? This figure is somewhat hard to determine because of the uncertainty at this time of the definition of pediatric hypertension. Taking into account the variations in methods of study and in measurement techniques in 16,000 children from various authors throughout the country, prevalence of hypertension has varied from 2-12% as compared to an adult figure of 20%. With this relatively remarkable prevalence in children, one cannot help but agree that children should have their blood pressure taken during office visits. Loggie suggests this be done in all children greater than three years old.

The technique of blood pressure measurements in children is important. One should use a cuff which has a bladder which will cover two-thirds of the upper arm. The cuffs bladder's length should totally encircle the patient's arm. More refined and newer techniques which show more accuracy in blood pressure determinations are the doppler pressure measurement which approaches arterial line pressure determination with considerable accuracy and a random zero sphygmomanometer which the British are using.

Below are some conditions which may cause persistent or transient hypertension in childhood:

Remediable Causes of Persistent Hypertension

I. Coarctation of the thoracic or abdominal aorta

II. Renal

1. Renal artery abnormalities (stenosis, arteriopathies, fibromuscular dysplasia, thrombosis, neurofibromatosis, fistula, aneurysm)
2. Renal vein thrombosis
3. Unilateral dysplastic kidney
4. Unilateral hydronephrosis
5. Unilateral pyelonephritis
6. Unilateral multicystic kidney
7. Traumatic damage e.g., constrictive perirenal hematoma
8. Solitary renal cyst
9. Renal tumors
10. Unilateral ureteral occlusion
11. Ask-Upmark kidney
12. Vascular or unilateral renal parenchymal disease following irradiation.

III. Adrenal

1. Neuroblastoma
2. Pheochromocytoma
3. Cortical hyperplasia (adrenogenital syndromes)
4. Cushing's disease
5. Primary aldosteronism (hyperplasia or adenoma)

IV. Miscellaneous

1. Ingestion of excessive amounts of licorice
2. Chronic administration of glucocorticoids, oral contraceptives, testosterone
3. Intracranial tumors

Incurable Causes of Persistent Systemic Hypertension

I. Surgically irremediable coarctation of the aorta or generalized hypoplasia of the aorta

II. Surgically irremediable renal artery abnormalities

1. Bilateral congenital dysplastic kidneys
2. Bilateral hydronephrosis
3. Chronic bilateral pyelonephritis
4. Polycystic kidneys
5. Medullary cystic disease
6. Chronic glomerulonephritis (all forms including those due to connective tissue disease)
7. Post-renal transplantation
8. Bilateral renal parenchymal damage from irradiation

III. Adrenal

1. Disseminated neuroblastoma
2. Inoperable or metastatic pheochromocytoma
3. Adrenal carcinoma

IV. Miscellaneous

1. Essential hypertension
2. Lead nephropathy (late)
3. Familial dysautonomia (intermittent hypertension)
4. Intracranial tumors (partially treatable or non-treatable)

Entities Associated with Transient or Intermittent Hypertension

I. Renal

1. Acute poststreptococcal glomerulonephritis
2. Hemolytic-uremic syndrome
3. Anaphylactoid purpura with nephritis
4. After genitourinary tract surgery
5. Renal calculus with colic
6. After renal transplant
7. After blood transfusion in azotemic patients
8. Anephric hypervolemia

II. Adrenal - corticosteroids

III. Miscellaneous

1. Oral contraceptives

2. Raised intracranial pressure (any cause)
3. Pre-eclamptic toxemia of pregnancy
4. Burns
5. Guillain-Barre syndrome
6. Hypernatremia
7. Hypercalcemia
8. Leukemia
9. Poliomyelitis
10. Mercury poisoning
11. Amphetamine overdosage
12. Reserpine overdosage (ingestion in young children)

Until recently essential or primary hypertension was considered a rare entity in children. In 1969 a study by Loggie demonstrated that only 20% of pediatric hypertensive patients had essential hypertension. In 1975 the percentage rose to 55%. This remarkable change in distribution may well reflect the increased awareness of primary care physicians who are now cognizant of the scope of the problem of pediatric hypertension, and routinely do blood pressures on their children in an office sitting. It is unclear how to manage the patient with borderline hypertension at this time. A Veterans Administration cooperative study was unable to show conclusively that treatment significantly altered the prognosis of adult patients with diastolic pressures between 90 and 105 mm. of mercury. This lack of definitive conclusions may have been due to the limited duration of the study.

It is probably wise to follow patients periodically with borderline hypertension and graph their blood pressures against the percentiles for age in order to evaluate their blood pressure trends. Since obesity and hypertension have a known association, we should encourage weight loss in patients with borderline hypertension who are obese and suggest that they should avoid excessive salt ingestion. Teenagers with borderline hypertension should be counselled against smoking and female patients about the possibility of oral contraceptive agents as an aggravating factor in hypertension. A good history and physical examination are mandatory in borderline hypertension patients, but laboratory examination in this population need not be extensive. In one study of 41 patients ages 12-13, who had borderline elevations of blood pressure, 20 intravenous pyelograms done were normal and 20 metanephrine levels were within normal limits. Chemical studies for electrolytes, blood urea nitrogen, calcium, and urinalysis were also normal in these patients. It is important to remember that the laboratory investigation in patients with borderline hypertension must be guided by the history and physical examination. The same can be said for patients whose blood pressure is above the 95th percentile. Excluding black patients, essential hyperten-

sion is quite rare under the age of 10. In these patients a more extensive workup may be required.

In every patient with definitive hypertension at the University of Cincinnati the following studies are obtained: complete blood count, urinalysis, electrolytes, blood urea nitrogen, creatinine, uric acid, tryglycerides, cholesterol and lipoprotein electrophoresis. Urine cultures are taken in males and females with underlying renal disease. Creatinine clearance is also collected in adolescent patients. The following studies are not routine and are only obtained when history and physical so indicate: immunoelectrophoresis, fluorescent antinuclear antibody, calcium, phosphorus, glucose, total protein. This is based on eight years of experience with these tests. Electrocardiograms are unproductive, being negative in 75% of children with persistent hypertension. Chest x-rays are even less sensitive showing abnormalities in only 12% of children. Intravenous pyelograms, at this point, have not been fully evaluated in children. It is Loggie's impression that routine sequence intravenous pyelogram will give more information than the rapid sequence intravenous pyelogram. Renal biopsies are only done in patients with findings suggesting glomerulonephritis or arteriopathy.

More sophisticated procedures such as metanephrine, VMA, aldosterone, 17 hydroxysteroids and 17 ketosteroid determinations are only done on specific indications and physical examination. In a study from Cincinnati in patients ages 2 to 20 with persistence of systemic hypertension, the percentage of abnormal results with the above tests is well below 10%. Abdominal angiograms are much more likely to be helpful in patients from the age of 2 to 12 as compared to 12 to 20. This is also true of the intravenous pyelogram. I will note that pheochromocytomas do tend to be extra adrenal in origin in children and that abdominal aortography is mandatory for localization in these patients. Also it should be noted that 90% of children have sustained hypertension with pheochromocytomas as compared to a much lower percentage in adults.

As I have already made my diagnosis, I will go on to discuss the entity of coarctation of the aorta. There is some controversy but the entity was described by either Meckel or Paris over 200 years ago. Crafoord and Nylin in Sweden reported successfully operating on the first patient with coarctation in 1945. Gross and Hufnagel confirmed the feasibility of surgical correction in 1945 in this country.

There is no clear cut mode of inheritance and the incidence of coarctation of the aorta between males and females has varied from equal to male pre-

dominance. In older patients with isolated coarctation, males have an almost 2 to 1 predominance. It should be noted that there is an 8% incidence of associated congenital anomalies in patients with coarctation. These anomalies include patent ductus arteriosus, ventricular septal defect, cerebral berry aneurysms as well as aortic stenosis and insufficiency. The frequency of bicuspid aortic valve in patients with coarctation of the aorta has ranged from 13 to 85%. In a recent study of patients with coarctation echocardiography revealed that 53% had this abnormality. Another autopsy study showed an incidence of 32%. Noncardiac malformations are observed in at least 7% of patients with coarctation. These defects include those of Turner's syndrome, hypospadias, club foot and ocular defects.

The prevalence of coarctation from birth to 15 years of age has been reported to be about 1 in 12,000. It ranks about 5th or 6th among congenital cardiac defects. The usual site of the narrowing of the aorta is just distal to the origin of the left subclavian artery. Rarely coarctation is found in the abdominal and thoracic aorta. Microscopic examination shows a diaphragm-like effect composed of media as well as thickened and fibrotic intima at the sight of narrowing. Beyond the lesion there is post stenotic dilatation and a jet lesion on the aortic intima.

When coarctation is diagnosed in infancy, there is a high percentage of other congenital anomalies. These include patent ductus arteriosus, ventricular septal defect, atrial septal defect, and even relatively uncommon problems such as transposition of the great vessels and endocardial fibroelastosis.

The left ventricle becomes hypertrophic in coarctation because of the increased afterload. Collaterals produce a variety of signs, including dilated tortuous intercostal vessels which may produce characteristic notching of the ribs on x-ray. Incidentally, this finding was not present in this case, although it often is in a patient of this age. Dilated, pulsating subclavian or internal mammary and scapular arteries show the well developed collateral circulation often found in these patients. This must be evaluated prior to surgery to estimate the degree of the compensation for the defect by collateral circulation.

Although patients who are older often present with hypertension; infants with coarctation, particularly in the first three months, may present with heart failure and in critical condition with cyanosis, dyspnea, and tachypnea. Examination of the heart reveals a prominent heave in the left anterior chest wall. There is a systolic ejection murmur present best heard along the left sternal border which is

often of grade II to IV (six grades) intensity. The murmur may also be heard in the back. The characteristics of this murmur may be influenced by associated heart defects. It is important to remember that some infants with severe failure may have no murmurs. The upper extremity pulses are much stronger than the lower extremity pulses. There is a gradient of over 20 mm of mercury of the upper extremities over the lower extremities in most cases of coarctation. There are two situations in infants in which this gradient of blood pressure between the upper and lower extremities does not occur: 1) When the patient is in severe left ventricular failure and the left ventricle is not able to generate sufficient pressure. 2) When the patient has a large ventricular septal defect or patent ductus arteriosus. Although the electrocardiogram in infants usually shows right ventricular hypertrophy and right axis deviation, this converts to left ventricular hypertrophy as time goes on. The chest x-ray may demonstrate increased heart size and enlarged pulmonary vessels. Cardiac catheterization studies have shown that 90% of infant patients present with congestive heart failure and a great majority also have pulmonary hypertension.

It has been shown that medical and surgical treatment is markedly affected by the presence or absence of other congenital heart defects. If the patient has other associated defects, as mentioned above, survival after surgery is much higher than with medical treatment. In regard to surgery, the major disadvantage in doing surgery for coarctation in infants is a high recurrence rate. If at all possible, surgery is probably best performed after the age of 4, when the aorta reaches 50% of the adult size.

What symptoms are present in older children with coarctation? Often these children are asymptomatic. If the blood pressure is determined, it is elevated in the upper extremities and lower in the lower extremities. This can often be determined by simply feeling the difference in pulses between the two extremities. Electrocardiograms in the older age group are normal in over 50% of patients. Ten of sixty patients have evidence of left ventricular hypertrophy.

Findings on chest films have been emphasized. The inverted 3 sign is made by the ectatic left subclavian above the defect and the post stenotic dilatation below the coarctation of the aorta. (see Figs. 1 and 2)

Rib notching is rarely seen in the first year of life but is commonly found after age 5 in the posterior 4th to 8th ribs. The heart may or may not be enlarged on x-ray. The frequency of cardiac defects such as patent ductus arteriosus, atrial septal



Figure 1

Marked narrowing at the area of coarctation of the aorta just distal to left subclavian artery.

defects, and ventricular septal defects, as well as pulmonary hypertension is much less common in children than in infants. The most common abnormality in children is a deformed aortic valve resulting in stenosis or insufficiency. Deformed valves were noted in 35 of 52 patients who had angiograms of their ascending aorta.

What is the course and natural history of untreated coarctation? Campbell in 1970 calculated the mortality rates above 30% in autopsy series in three separate clinical studies totaling 161 patients. Of patients surviving the first two years of life, 25% died before age 20, 50% before age 32, 75% before age 46 and 90% before age 58. The mean age of death was 34 years. Congestive heart failure was very frequent in patients under one year or over 30 years of age.

There are four major complications in coarctation. These include: 1) congestive heart failure, 2) rupture of the aorta or dissecting aneurysm of the aorta, 3) bacterial infection of bicuspid aortic valves or the area of coarctation, and 4) cerebral vascular hemorrhage, often due to ruptured berry aneurysm. Bicuspid aortic valves often become fibrotic and calcific forming the basis of aortic stenosis in adults.

Because of the shortened life span and frequency of complications, surgical correction of coarctation of the aorta is recommended. As I have suggested before, because the aorta enlarges as a child grows, elective surgery should be postponed until after four years of age to avoid the possibility of recurring coarctation. Surgical mortality is higher in infants because of the associated cardiac lesions and in patients over thirty years of age because of friability of the aorta. Surgical mortality in the four to five year age group is from 0-5% but after thirty years of age rises to 6-13%.



Figure 2

Inverted "3" sign in coarctation with narrowing and post-stenotic dilatation.

There are several postoperative complications. One is mesenteric arteritis which was present in 14% of 1,193 in surviving post-surgical patients. The mesenteric arteritis is preceded by paradoxical hypertension. This complication is seen five times more commonly in patients under twenty years of age. If untreated, bowel necrosis secondary to the arteritis may occur. The mortality of this complication is 9%.

Recurrence of coarctation occurs in 34% of patients operated in the first year of life and in 9% of patients of 1-5 years of age but only in 2% of patients operated on after the age of five. Reoperation is indicated for patients whose pressure differential between the upper and lower extremities exceeds 40 mm. of mercury or in whom the diameter of the aorta at the point of coarctation is less than 55% of the remainder of the aorta. In long term follow-up in 194 patients, who underwent repair of the aortic coarctation (excluding patients operated before age two) and followed for 11-25 years; there was a high incidence of premature cardiovascular disease. Seventy percent of patients had aortic valve disease (presumably related to bicuspid aortic valves). In 190 patients whose mean age at surgery was 15 years of age the mean age at death was 32.5 years. Persistent hypertension has ranged from 7-25% in various studies.

Dr. Stassen's Diagnosis:

Coarctation of the Aorta with Possible Bicuspid Aortic Valves

DR. BARLOW: I would like to ask Dr. Sullivan to make some comments as he detected hypertension in this patient on a routine office visit for allergic rhinitis.

***DR. CHARLES SULLIVAN:** This patient had blood pressures which were up to 150/90 requiring diazide therapy. After the resection of the coarctation of the aorta by Dr. Reynolds, the patient still remained somewhat hypertensive postoperatively but at this time is doing very well with a systolic blood pressure of 95-100 and a diastolic blood pressure of 75-80 mm of mercury. He has gained one inch in height. Although the patient denied headache, decreased exercise tolerance, or claudication in the legs prior to surgery, he says that he does feel better after surgery and has increased exercise tolerance although he did not note any problem prior to surgery.

Because this child had some webbing in the neck and was short, we did draw blood and obtain skin fibroblasts for chromosome analysis. I have no report on these studies as yet. I will note that both on aortogram and on echocardiography, he did not appear to have a bicuspid aortic valve.

DR. REYNOLDS: I would like to compliment Dr. Sullivan for his persistence in evaluation of asymptomatic hypertension in this patient. The chest films in this patient do not show rib notching or remarkable abnormalities. However, I will show films of his aortogram which were performed through the right brachial artery approach. Views of the arteriogram show three cusps of the aortic valve. In the area after the origin of the left subclavian artery in the proximal aorta, the coarctation and the post-stenotic dilatation form the so-called inverse 3 sign (Figs. 1 and 2). One may occasionally see this sign not only in the arteriogram but also on a barium swallow where the esophagus is indented by the segments of the aorta that I mentioned.

Classifications of coarctation of the aorta often utilize the location of the narrowed segment. Pre-ductal, juxtaductal and postductal locations are terms often used. This classification is analogous to the previous classifications which include the pre-ductal or infantile type, which is associated with a high incidence of cardiac anomalies and has a poor prognosis in contrast to the more common adult or postductal type of coarctation seen after infancy. Often juxtaductal and postductal are lumped together. This patient today fits into this postductal adult category.

I want to again emphasize the importance of detecting the patient with asymptomatic hypertension.

The patient otherwise may be first seen after he had developed one of the complications such as dissection of the aorta or intracranial hemorrhage. Dr. Stassen has discussed these complications nicely. Surgical therapy has been fairly well delineated. The infantile group is more complex because of the associated cardiac anomalies. These infants tend to be extremely ill and their mortality can be as high as 35-40%. If one excludes this early age group from the surgical series, several series have reported surgical mortality rates in the range of 1-3%.

These patients do not usually require cardiopulmonary bypass but sometimes a patient will not tolerate clamping of the aorta so the surgeon must be prepared to perfuse the lower portion of the body. In this patient we measured pressures in the lower aortic segment after the aorta had been cross-clamped. As the pressure differential before and after cross clamping was only about 5 mm of mercury, perfusion of the distal aorta was not necessary. In this patient we were able to resect the narrowed aortic segment and perform an end-to-end anastomosis. This is the preferred technique. If there is a long coarcted segment or a long area of aortic hypoplasia, a dacron graft may have to be employed. Occasionally one may turn the left subclavian artery into the distal aorta. Dr. Stassen has nicely pointed out the problem of recurring coarctation which is a problem limited primarily to infants. Excluding this group surgery should be performed soon after the diagnosis is made.

The mesenteric arteritis seen as a postoperative complication of coarctation surgery is poorly understood. It has been felt that the mesenteric arteries have not been previously subjected to a pulsatile flow and develop arteritis which may produce necrosis of the bowel. We are slow to start these patients on oral fluid postoperatively under the assumption that the bowel must adjust to the new arterial flow. It is interesting to note that you can often determine the extent of collateral circulation by pulsations in the intercostal vessels or in the subscapular arteries in the latissimus dorsi. Occasionally, if the coarctation is located before the origin of the left subclavian, one may note striking pulsations in to the right latissimus dorsi but none in the left.

****DR. ROBERT WILLIX:** As has been suggested, perfusion of the kidneys and lower aorta is extremely important in these patients to prevent complications. One of the most serious of these, of course, is paraplegia secondary to ischemia to the spinal cord. Paraplegia has occurred with or without cardiopulmonary bypass or shunts. This complication of paraplegia is of great concern to us, particularly in

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this age group where the mortality of surgery may be as low as 1%. In older patients where the aorta is more friable, surgical mortality rises.

One way of protecting these children from possible paraplegia is what was done in this case. One gains control of the aorta above and below the coarctation with as little dissection as possible so as not to impair any circulation to the spinal cord. The pressure should then be measured in the distal stump; and, if there is a perfusion pressure of 50 mm of mercury or over, this should be adequate. There is no need to perform a shunt to supply the kidneys and lower extremities. If the perfusion pressure is below 50 mm of mercury, one can shunt blood to the distal aorta by two methods. In the first, a heparin bonded plastic shunt (Gott shunt) is used to bypass the coarctation. The second is the use of limited cardiopulmonary bypass, thereby supplying blood from the left atrium to the femoral artery. This is a left heart bypass in which blood is supplied to the lower extremities by the pump and to the brain and upper portion of the body directly from the heart.

I would also like to mention that if a patient has asymptomatic hypertension and an angiogram is performed, the coarctation may not be demonstrated if only the usual location is investigated. Coarctations of the abdominal and descending thoracic aorta do occur but they are rare.

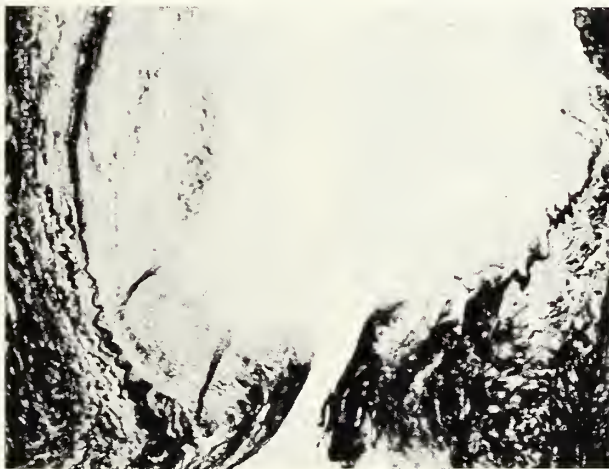


Figure 3

Section through area of coarctation shows marked narrowing of lumen by fibrous intimal proliferation (see text).

DR. BARLOW: This is a photomicrograph of the coarcted segment of aorta. The small, almost pinpoint lumen in the center represents the only passageway for blood. (Fig. 3). The elastic tissue stain shows some characteristic changes. The aortic media extends like a curtain across the aorta. Toward the small central lumen one can see a lighter staining tissue which represents fibrosis of the intima. This

fibrosis is thought to be secondary to the buffeting effect of the blood as it traverses the coarcted segment. Therefore, one would suspect that coarctations really can become more severe with time. (Fig. 3)

ANATOMIC DIAGNOSES: COARCTATION OF AORTA, ADULT, POSTDUCTAL TYPE

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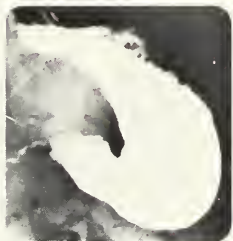
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King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

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Additional methods include the use of positive and negative control sera with serological procedures; the use of known types of bacteria to verify

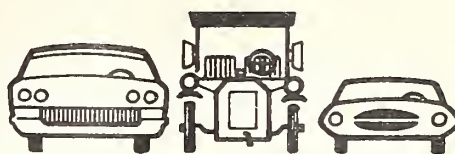
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the sensitivity reactions to various antibiotics, to validate the quality of bacteriological media, and to check gram stains and acid-fast stains; and the use of controls for hematology determinations such as hemoglobin, hematocrit, leukocyte counts and erythrocyte counts. Blood banks check the specificity and potency of commercially prepared antisera against known cells on a daily basis usually.

The attending physician plays a key role in quality control by requesting repetition of laboratory work that appears to conflict with the clinical status of the individual patient. In addition, **external quality control** consists of laboratory participation in proficiency test programs conducted by outside, independent organizations such as the College of American Pathologists, the Center for Disease Control, the American Association of Blood Banks, and state health laboratories (primarily for syphilis serology). In such programs unknown specimens are submitted to the laboratory which are to be treated in the same manner as specimens from patients. The results of these tests are then submitted to the sponsoring organization which in turn issues a report containing a comparison of the results obtained in a given laboratory with those from reference laboratories (which serve as the referees) and other participating laboratories on a nationwide basis. Where possible the laboratory is scored for each test, and is compared with other laboratories that use the same equipment and methods.

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Lecture #11

Preeclampsia

Loren P. Petersen, M.D.*

DEFINITION

Preeclampsia is the development of hypertension during pregnancy, or subsequent to a recent pregnancy. Edema and/or proteinuria are frequently associated with pregnancy induced hypertension; however, hypertension is necessary to establish the diagnosis of preeclampsia. Hypertension during pregnancy is defined as a rise in the systolic pressure of at least 30 mm Hg, or a rise in the diastolic pressure of at least 15 mm Hg, or the pressure of a systolic pressure of at least 140 mm Hg, or a diastolic pressure of at least 90 mm of Hg.

Gestational edema occurs in approximately 60% of normal pregnancies. Excessive weight gain (>1 Kg/ week) is found in nearly all patients with preeclampsia; however, edema by itself should be considered normal during pregnancy, and does not require either low salt diet or diuretic therapy.

Proteinuria is the presence of protein in concentrations greater than 0.3 gm/liter in a 24 hour urine collection, or greater than 1 gm/liter (1+) in a random urine collected on two or more occasions, at least six hours apart. The proteinuria in preeclampsia contains albumin, transferrin, and IgG. In addition, the principal biochemical changes following heavy proteinuria in preeclampsia are identical to those found in the nephrotic syndrome (decreased maternal plasma concentration of albumin, transferrin, and IgG, a two-fold increase in alpha 2 macroglobulin, and a ten-fold increase in the concentration of beta lipoprotein).^{1,2}

Several attempts have been made in order to classify hypertensive disorders during pregnancy. The following classification is clinically helpful in managing patients with hypertension and pregnancy:

Preeclampsia: (Gestational induced hypertension)

Mild: BP 140/90

Edema

No proteinuria

Moderate: BP 140/90 - 160/110

Edema

Proteinuria

Severe: Diastolic blood pressure >110

Proteinuria >5 gms/24 hours

Oliguria <500 ml/24 hours

Pulmonary edema

Abnormal liver function

Thrombocytopenia

Disseminated intravascular coagulation

Eclampsia: Convulsions or coma in a patient with preeclampsia.

Superimposed Preeclampsia or Eclampsia: The development of eclampsia or preeclampsia in a patient with chronic hypertensive or vascular disease.

Chronic Hypertensive Disease: The presence of persistent hypertension for whatever cause, before pregnancy or before the 20th week of gestation, or persistent hypertension beyond the 42nd post-partum day.

Unclassified Hypertensive Disorders: Those disorders in which information is insufficient for classification.

Preeclampsia is almost exclusively a disease of

*Professor and Chairman, Department of Ob/Gyn, University of South Dakota School of Medicine, Yankton, South Dakota

the primigravida. When preeclampsia occurs in the multipara, it is usually superimposed on hypertensive vascular disease or associated with other predisposing factors such as hydramnios, multiple pregnancy, chronic renal disease, diabetes mellitus, or hydatid mole. In addition, preeclampsia rarely occurs before the 20th week of gestation, is more common among the poor and those with suboptimal nutrition, becomes more prevalent as term approaches, and disappears when the uterus is empty. The reported incidence varies from .5 to 30% because of differing criteria for diagnosis, antenatal care, general health of the community, race, and area of the world being studied.

Prenatal Predictability of Preeclampsia: Normotensive pregnant women are resistant to pressor effects of exogenously administered angiotensin II. These observations led Gant to study prospectively pregnant women with angiotensin II infusions and to reliably predict which patients would develop preeclampsia. In 1974, Gant et. al. reported on a clinical test (Roll Over Test) for predicting the development of preeclampsia. The patient's blood pressure (at 28-32 weeks gestation) is taken in the lateral recumbent position until a baseline BP is established. The patient is instructed to roll over on her back, at which time the blood pressure is taken immediately, and then 5 minutes later. If the diastolic pressure increased by 20 mm of mercury, the test is considered positive. Gant reported 93% predictability for a positive test. Controversy remains regarding the reliability of the test; however, at least some information is obtained which is helpful in selecting which patients to closely follow for preeclampsia.^{3,4}

The major complications of severe preeclampsia include:

Abruptio Placentae: Twenty five percent of patients with abruptio placentae have pregnancy-induced hypertension.

Altered Clotting Mechanisms: One of the earliest findings of disturbed clotting mechanisms is thrombocytopenia. Twenty three percent of cases with post-partum hypofibrinogenemia were associated with pregnancy-induced hypertension.

Cerebral Hemorrhage: The most frequent cause of maternal death.

Hepatocellular Damage: Seen frequently in severe disease, and may be monitored by enzyme determinations.

Glomeruloendotheliosis: Present in 75 percent of patients who have a clinical diagnosis of preeclampsia, and are primigravida.

Fetal complications seen in moderate and severe preeclampsia include: prematurity, intrauterine growth retardation, stillbirths, and intrapartum deaths. Fetal evaluation is an integral part of management of the preeclamptic pregnant patient. Fetal evaluation should include frequent estriol determinations, OCT testing, amniocentesis, and fetal monitoring during labor. Ultrasound is helpful in moderate and severe preeclampsia in order to rule out intrauterine growth retardation.

Management of Mild Preeclampsia: The objective of therapy in mild preeclampsia is to prevent progression of the disease. *Diuretics* have little or no place in the treatment of mild, moderate, or severe preeclampsia, and they are potentially harmful to the fetus (thrombocytopenia and electrolyte imbalance). Preeclampsia is a hypovolemic state. Diuretics further reduce blood volume, decrease placental exchange (diminished metabolic clearance rate of dehydroisoandrosterone sulfate: Gant, et. al., 1975), and have no effect whatsoever on the course of the disease. Occasionally, diuretics may be indicated for postpartum oliguria.

For mild preeclampsia, hospital admission is necessary to rule out a more severe, unsuspected form of the disease. Bed rest normally decreases aldosterone secretion, allows natriuresis, and reduces both plasma renin and angiotensin II. Bed rest is often adequate treatment. Houth et. al. treated 346 nulliparous women with pregnancy-induced hypertension in the hospital, at bed rest, with a regular hospital diet without salt restriction, no diuretics, and no sedation. The perinatal mortality rate was 9/1000, and the maternal mortality was zero.^{6,7}

The use of phenobarbital in mild preeclampsia has three advantages: (1) Phenobarbital given to the mother for 10 days prior to delivery will induce fetal liver glucuronyl transferase, and help prevent neonatal hyperbilirubinemia. (2) It is an anticonvulsant. (3) The mild sedation helps patients maintain bed rest. Therapy of mild preeclampsia is thus: bed rest, good nutrition, no salt restriction or diuretics, and mild sedation with phenobarbital. If the patient's condition is stable, one can stop the phenobarbital 3-5 days prior to delivery, and hopefully, reduce the neonatal sedative effects. The decision to deliver the patient with mild preeclampsia is determined by gestational age (delivery after 37 weeks), non-stress testing, estriol levels, and mature amniotic fluid L/S ratios. Ultrasound is helpful in order to rule out intrauterine growth retardation.

Management of Moderate and Severe Preeclampsia: The essential objectives of management are to deliver a live baby, without damage to the mother.

Evaluation of the extent of the maternal pathological process is as essential for deciding when to end the pregnancy, as is fetal evaluation. Frequently, the maternal condition is not adequately evaluated, and the disease progresses to renal failure, hepatic destruction (liver rupture), or disseminated intravascular coagulation, and the whole process has been unrecognized. Maternal evaluation should include the following as minimum diagnostic evaluation:

- (1) Frequent evaluation of vital signs, BP, levels of consciousness, reflexes, and hourly intake and output.
- (2) Daily or every other day evaluation of clotting studies, especially the platelet count. Thrombocytopenia is one of the first signs in toxemia of impending generalized DIC.^{8,9}
- (3) Liver function studies, SGOT, LDH every 2-3 days. The SGOT gives important information regarding hepatocellular damage.¹⁰
- (4) Renal function, creatinine clearance, BUN at least 2-3 times weekly. A decreasing creatinine clearance indicates increasing severity of maternal disease.
- (5) Daily or every other day monitoring of the hematocrit, hemoglobin, uric acid, and the electrolytes—especially if diuretics have been given.¹⁰
- (6) EKG will give information regarding myocardial ischemia, and is vital in patients with severe preeclampsia and Class B-F diabetes mellitus.

Immediate fetal evaluation should include:

- (1) Ultrasound
- (2) Amniocentesis L/S ratio
- (3) Serum Estriol or Estetrol (daily)
- (4) Serum HPL
- (5) Non-stress fetal monitoring (or preferably oxytocin stress testing).

Therapy of moderate and severe preeclampsia is three-fold: $MgSO_4$, control of blood pressure, and termination of the pregnancy.

Magnesium sulfate: The drug of choice in order to prevent preeclampsia. The standard intravenous dose is 2.0 to 3.0 gms of a 10% solution given *slowly* (over at least 20 minutes), and 1.0 gm per hour by slow intravenous infusion with frequent (every 15 minutes) monitoring of urine output, reflexes, BP, and respirations. A bolus of 2.0 gms $MgSO_4$ given IV has been shown to produce respiratory changes in 79% of patients, including shallow and slow respirations to complete apnea. Therefore, a bolus of $MgSO_4$ rapidly injected should be avoided.¹¹

Blood pressure: Best controlled with hydralazine (Apresoline) by intravenous drip in the acute severe

hypertensive crisis. Maintaining the diastolic pressure below 110 is the specific aim of therapy. Apresoline probably increases utero placental blood flow, and is, therefore, of theoretical benefit to the fetus.

Termination of the pregnancy: In moderate or severe preeclampsia, is definitive, and should be carried out as soon as possible with any of the following:

- (1) Mature L/S ratio
- (2) Gestational age > 37 weeks
- (3) Abruptio placentae
- (4) Late decelerations on OCT
- (5) Falling estriol levels
- (6) Uncontrolled blood pressure, regardless of fetal status
- (7) DIC

Strong consideration for immediate delivery should be given when any of the following are present:

- (1) Decreasing urine output
- (2) Decreasing creatinine clearance, or increasing BUN
- (3) Increasing SGOT
- (4) Falling platelet counts
- (5) Increasing uric acid
- (6) Prolonged (> 24 hours) $MgSO_4$ therapy necessary to control reflexes and prevent eclampsia.

The decision of whether induction of labor or to perform a cesarean section is reached on the basis of fetal monitoring heart rate patterns, severity of maternal disease (expediency for delivery), fetal scalp pH, etc.

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1. Is the meeting room conveniently located and in a quiet area? Are adjacent meeting rooms used by others? Are foyers heavily traveled? Are elevators or stairs near the meeting room?
2. How soundproof is the room? This is particularly important when sharing part of a large room that is divided by a temporary partition. If another meeting is being held simultaneously, what effect will the use of audio equipment have?
3. Are directional signs needed? In hotels, utilize the hotel lobby directory.
4. Place a sign outside the meeting room, "Quiet—Meeting in Progress." Also, identify the meeting being held. This will lessen the number of uninvited guests walking in on the session.
5. Check to see that the room is well ventilated and lighted. Specify the desired temperature. Know beforehand where all light switches and outlets are located.
6. Know the name and phone extension of the engineer on duty, in case of any emergency with lighting, sound or ventilation.
7. Determine what seating arrangement is best suited for the group. There are a number of arrangements, but have the committee seated so the chairman and members can easily see and talk to each other. Triangular and closed square arrangements are good. Depending on the size of the committee and other needs, a circular open square, or U-shaped arrangement is practical. The key consideration is to provide good contact and rapport among all members.
8. Chairs should be comfortable—particularly important for lengthy meetings.
9. Provide ashtrays, water and glasses, note pads and pencils.
10. Use name cards on table in front of each member.
11. Know location of checkroom and rest rooms.
12. If a chalkboard is to be used, have eraser and chalk available.

13. Test all electrical equipment.
14. If film or slides are to be used, check for proper operation of projector and screen in advance.
15. If visual aids are used, place them for convenient viewing. Avoid the rearrangement of chairs during the meeting.
16. Order sufficient beverages for coffee breaks. Not everyone drinks coffee, so have other refreshments available.
17. Have lectern and gavel available.
18. Have extra copies of the agenda and other supplementary material available.

SDAFP Memorial Lecture

The SDAFP Board of Directors, meeting during the 1978 Black Hills Summer Seminar, acted favorably upon a recommendation from the Education Committee and has sanctioned the development of an "SDAFP Memorial Lecture" to be given at each of the chapter sponsored Black Hills Seminars, beginning with the 1979 Black Hills Summer Seminar.

This lecture is to be given by an active, practice affiliate or resident affiliate member of SDAFP on a topic of the speaker's preference. Suggested topics are available from the Education Committee, based upon the cyclic core of knowledge for family practice.

Member applications for the privilege of being selected for this lecture must be available to the state office by April 1 of each year for the Summer Seminar and October 15 for the Winter Seminar. The application will be a letter of intent to be selected and an outline, with references, of the proposed lecture, including the title. The speaker selected for each of these lectures will be handled by the SDAFP Education Committee through a review process.

This memorial lecture, dedicated to former SDAFP members now deceased, will carry the honorarium award of \$200. Your participation is invited.

From PURE GOLD by Ole E. Rolvaag

To all men is portioned out a fair share of adversity. There is only this difference: some get more than others and some stand up bravely under it; others are broken utterly, and are swept away like chips upon a swift current. But mountain peaks crumble and high hills are worn low, so why should the poor earthlings crawling upon them complain?

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SD

This Is Your Medical Association

Rodney Parry, M.D., Assistant Professor of Medicine, USD, presented a workshop on chronic obstructive pulmonary disease during the annual meeting of the South Dakota Lung Association held in Mitchell.

* * * *

L. C. Askwig, M.D., and **Robert Hayes, M.D.**, Pierre, and **N. R. Whitney, M.D.**, Rapid City, were elected to the medical advisory committee of the Crippled Children's Hospital and School in Sioux Falls.

* * * *

The South Dakota Chapter of the American Academy of Pediatrics received the 1978 Wyeth Outstanding Chapter Award for outstanding achievement. This award which was presented to only four chapters this year was accepted by **N. R. Whitney, M.D.**, Rapid City, South Dakota Chapter president, during the annual business meeting in Chicago.

* * * *

A paramedic training program for ambulance drivers has been established in Pierre with **M. R. Cosand, M.D.** serving as medical director. Other physicians teaching in this program include **R. C. Jahraus, M.D.**, and **R. J. Zakahi, M.D.**, Pierre, and **Barry Pitt-Hart, M.D.**, Sioux Falls.

* * * *

Several South Dakota physicians participating in diabetes classes in their own communities included **Nick Guddal, M.D.**, Watertown; **William Taylor, M.D.**, Aberdeen; and **Steven Haas, M.D.**, Rapid City.

Paul Leon, M.D., Aberdeen, was featured speaker at the Diagnostic Procedures and Techniques Workshop sponsored by the South Dakota Practical Nurses Association held in Aberdeen.

* * * *

Jerome Eckrich, M.D., Aberdeen, was honored by Creighton University and the Creighton University Alumni Association for outstanding service. He was recognized for his fifty years of service and for his loyalty to Creighton University. He is a 1928 graduate of Creighton.

* * * *

Dean Madison, M.D., Sioux Falls, has been named a Fellow of the American College of Obstetricians and Gynecologists.

* * * *

Speakers for a seminar on infection control held in Rapid City included **J. T. Elston, M.D.**, **Gerald Butz, M.D.**, **Charles Loos, M.D.**, **William Howard, M.D.**, and **Myron Jerde, M.D.**, all of Rapid City.

* * * *

The Aberdeen Area Chapter of the American Association of Retired Persons heard a program on high blood pressure and related problems of stroke and heart disease presented by **E. P. D'Souza, M.D.**, Aberdeen.

* * * *

Steven C. Johnson, M.D. has joined **Drs. Mutch, Looby and Madison** in the practice of obstetrics and gynecology in Sioux Falls. Dr. Johnson graduated from the University of Iowa College of Medicine, interned at McKennan Hospital in Sioux Falls, and completed his residency training at the University of Iowa before returning to South Dakota.

* * * *

**YOUR
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Harold P. Henrie, M.D., Hot Springs, died at age 47. Dr. Henrie was a graduate of the University of South Dakota School of Medicine and received his M.D. degree from the University of Washington School of Medicine. He served a residency in psychiatry at the Veterans Administration Hospital, Perry Point, Maryland, and practiced in Rapid City and Hot Springs. Dr. Henrie is survived by his wife, Sharon, and five children.

SD

Letters To The Editor

This letter is in the form of a general congratulation to those students at the University of South Dakota who worked to make the Ethics Legalities and Economics of Medicine seminar held on November 1st, 2nd and 3rd so successful.

This seminar was about issues that each of us deals with on a day to day basis in our clinical practice, and I encourage the continuation of this program. I also encourage the attendance by other physicians, as I believe it to be one of the most outstanding programs that I have attended in the State of South Dakota.

I am pleased with the desire that was displayed by the students to learn about people, and to prepare themselves to go out and care for people—instead of taking care of people.

I encourage the continued support of seminars like these by the students, but I also encourage practicing physicians to participate.

Faternally,
Charles L. Pelton, M.D.

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CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, hypertensive crises may result.

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle, the patient should therefore be cautioned accordingly. **Drug Dependence.** Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychological dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. **Use in Pregnancy:** Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. **Use in Children:** Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: **Cardiovascular:** Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. **Central Nervous System:** Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache, rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. **Gastrointestinal:** Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. **Allergic:** Urticaria, rash, ecchymosis, erythema. **Endocrine:** Impotence, changes in libido, gynecomastia, menstrual upset. **Hematopoietic System:** Bone marrow depression, agranulocytosis, leukopenia. **Miscellaneous:** A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in the evening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg tablet daily, swallowed whole, in the morning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phenolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

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References: 1. Citations available on request—Medical Research Department, MERRELL RESEARCH CENTER, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon, R.H., and Leyland, H.M.: A Comprehensive Review of Diethylpropion Hydrochloride. International Symposium on Central Mechanisms of Anorectic Drugs, Florence, Italy, Jan. 20-21, 1977.

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Poor Correlation Of Symptoms, Roentgenograms And Pulmonary Functions In A Case Of Endobronchial Sarcoidosis

Jay Bachmayer, M.D.*

Louis W. Burgher, M.D., Ph.D.**

Abstract

A case of bronchostenotic endobronchial sarcoidosis serially evaluated by fiberoptic bronchoscopy, chest roentgenograms and pulmonary function tests is presented. The poor correlation of these parameters is noted and brief discussions of the value of endobronchial biopsy and transbronchoscopic lung biopsy in this entity are presented.

Fiberoptic bronchoscopy in conjunction with transbronchial lung biopsy has been found to yield tissue diagnostic of parenchymal sarcoidosis in up to 91% of cases.¹ Other authors have directed attention to endobronchial sarcoid involvement, the macroscopic findings ranging from normal mucosa to bronchial stenosis.²

Transbronchial lung biopsy and serial endobronchial biopsies were obtained in a patient with multiple bronchostenosis. The diagnosis of sarcoidosis was established on the basis of the clinical status, and confirmed histologically on the basis of the endobronchial biopsies.

CASE REPORT

A 43 year old non-smoking female was first evaluated in March, 1975 for multiple pulmonary infiltrates, a dry non-productive cough present for 10 months and dyspnea on moderate exertion.

On admission, physical examination revealed a healthy-appearing female, with decreased breath sounds over the right mid-lung field posteriorly. Routine, fungal, and acid fast cultures were negative. Serum allergic precipitins, mumps and interme-

diate PPD skin tests, and fungal serology for histoplasmosis were all negative. Chest roentgenograms revealed bilateral alveolar infiltrates, and bilateral hilar adenopathy. (Fig. 1A)

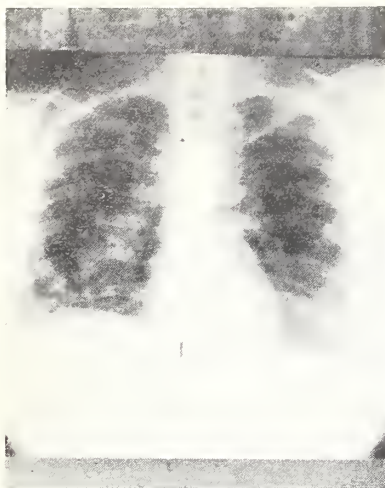
Pulmonary function testing (Table I) revealed restrictive as well as obstructive changes, and arterial hypoxemia. Fiberoptic bronchoscopy (FOB) was performed revealing pale white, opalescent masses obstructing the orifices of LB 2-3, RB 1 and RB 4-5. Neither the bronchial brush nor forceps could be advanced through these obstructions. (Fig. 2A) Cytology was negative for malignancy. Biopsies taken from the orifices of the involved segmental bronchi disclosed submucosal granulomatous inflammation characterized by scattered multi-nucleated giant cells and epitheloid-appearing histocytes. The diagnosis of sarcoidosis, Stage II, was made and the patient dismissed on no specific therapy.

She remained asymptomatic until one week prior to her second admission in March, 1976. At that time she was having increasing shortness of breath and non-productive cough.

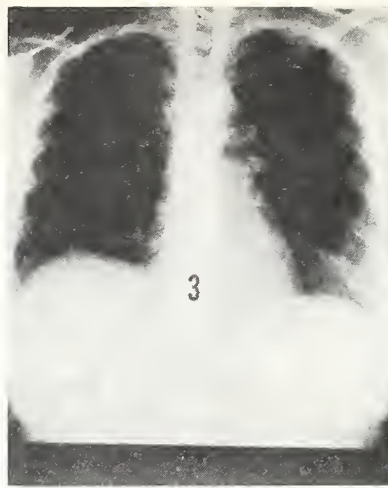
Physical examination revealed scattered wheezes and rhonchi throughout all lung fields. Chest roentgenogram revealed bilateral hilar adenopathy with progressive bilateral interstitial infiltrative changes of a linear-nodular type. Resting pulmonary function

*Department of Internal Medicine, University of Nebraska, College of Medicine, Omaha, Nebraska 68105.

**Medical Director, Respiratory Therapy Services, Bishop Clarkson Memorial Hospital, Omaha, Nebraska 68105.



(A) March 1975



(B) May 1976



(C) October 1977

Figure 1
Photograph of the serial chest roentgenograms

was significantly improved. (Table I) FOB demonstrated persistent obstruction of RB-1, RB-2 and RB 4-5 and LB 1-3 were all partially obstructed. Biopsy of the stoney hard obstructions demonstrated pathologic changes similar to those obtained one year ago. A transbronchoscopic lung biopsy showed pulmonary fibrosis. Because of the marked endobronchial involvement with stenosis the patient was started on prednisone 60 mg per day to be tapered over 3 months.

In May, 1976 FOB was repeated and revealed a normal bronchial tree on the left except for LB-3 which was stenotic but no longer occluded. RB 1 and 2 continued to be 90% stenosed. The remaining segmental bronchi continued to show involvement but were judged to be 20% improved. (Fig. 2B) Chest roentgenograms were significantly improved (Fig. 1B) while pulmonary functions were unchanged. (Table I)

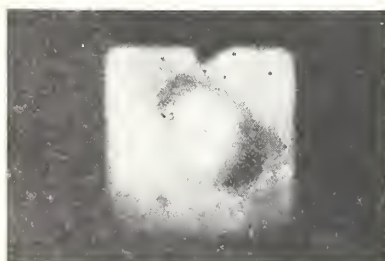
She was discharged on prednisone 40 mg on an alternate-day regimen. She was lost to follow-up until January 1977. She had complied with her alternate-day steroid therapy until June 1976 when she had discontinued her medication. She had re-

mained asymptomatic until December 1976 when she had increasing non-productive cough and shortness of breath. Her chest x-ray was unchanged from June 1976. Her prednisone therapy was reinstituted utilizing an alternate day regimen of 60 mg for one month followed by 40 mg Q.O.D. She returned in March 1977 asymptomatic. Her chest roentgenogram showed no interval change. She was instructed to reduce her prednisone to 30 mg Q.O.D. in one month. This could not be accomplished as she experienced increasing symptomatology on the lower dosage. She continued to be well clinically and showed some minimal radiologic improvement.

Clinical assessment in October, 1977 was unchanged. Repeat FOB demonstrated 80% patency of LB 1-3 but persistent obstruction of RB 1 and 2. RB 4-5 were now normal appearing. (Fig. 2C) Chest roentgenograms were essentially unchanged. (Fig. 1C)

DISCUSSION

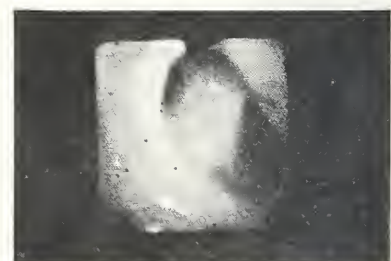
Since the initial isolated reports of histologic and macroscopic bronchial mucosal abnormalities, several authors have attempted to define the incidence of these abnormalities. This has varied according to



(A) March 1975



(B) May 1976



(C) October 1977

Figure 2
Photograph of serial endobronchial changes

patient selection and type of bronchoscope utilized. (Table II) Positive bronchial mucosal biopsies were reported in 43.4% (Stage I—31.8%, Stage II—57.3%, Stage III—45.5%). It should be pointed out that 34% of positive biopsies were from normal-appearing mucosa. These studies also suggest that

there is an increased incidence of biopsies positive for sarcoidosis if there are macroscopic changes (mucosal swelling, partial or total stenosis, parallel vessels, or yellow-white plaques), bronchial symptoms, or multiple biopsies. The incidence of bronchostenosis was 22.5%. The degree of bronchosten-

Table I
Pulmonary Function Tests Before and After
Treatment With Corticosteroids

	Predicted	2/75	3/76	5/76	10/77
TLC, liters	4.72	3.76	4.45	4.47	4.51
IVC, liters	3.01	2.64	2.55	2.60	3.17
RV, liters	1.71	1.12	1.90	1.87	1.35
RV/TLC, precent	36	29.9	42.0	41.0	29.0
FEV, liters	3.01	2.34	2.45	3.00	2.92
FEV ₁ , liters	2.26	1.64	1.45	1.85	1.96
FEV ₁ /FEV, precent	75	69	59	61	66
Peak Flow, liters/sec.	7.10	4.74	3.30	5.10	4.90
MEF 50%, liters/sec.	4.90	1.29	1.00	1.20	1.60
MEF 75%, liters/sec.	1.77	0.39	0.20	0.30	0.50
MVV, liters/min.	91.47	41.0	45.0	59.8	41.6
PaO ₂ , mmHg	80-100	49	87	84	82
PaCO ₂ , mmHg	35-45	39	32	29	36
pH	7.35-7.45	7.38	7.47	7.50	7.46
DL _{CO_{SB}}					
ml/min/mmHg	27.36	16.49	26.30	26.80	23.80
CV/VC%	15.70	21.0	13.11	13.33	15.6
Slope Phase III, % N ₂ /liter	1.43	1.49	2.52	1.59	3.11

Definition of abbreviations: DL_{CO_{SB}} = single breath gas transfer of carbon monoxide; CV = closing volume

Table II
Incidence of Positive Bronchial Mucosal Biopsies
And Bronchostenosis In Sarcoidosis

References	Cases Biopsied	Positive (%)	Stenosis (%)
I. Schiessle, et al. Munch Med Wschr 103:726-730, 1961	126	52%	NR*
II. Stahle Acta Med Scand Suppl. 425:234-36, 1964	72	26%	28.8%
III. Siltzbach, et al. Acta Med Scand Suppl. 425:230-233, 1964	49	61%	18.3%
IV. Carlens Acta Med Scand Suppl. 425:237-38, 1964	56	19.6%	21.4%
V. Friedman, et al. JAMA 183:646-650, 1963	35	62.8%	22.8%
VI. Turiaf Acta Med Scand Suppl. 425:228-229, 1964	71	49.3%	NR*
VII. Kalbian Thorax 12:18-23, 1957	10	30%	18.1%
Totals	419	42.9%	21.9%

*NR = not reported

osis often was not mentioned.

The clinicopathologic course of bronchostenotic sarcoidosis untreated with corticosteroids is poorly defined. Isolated case reports in the literature would suggest that without corticosteroids, bronchostenosis confirmed bronchoscopically or with bronchograms is a progressive process.^{3,4} Corticosteroid therapy seemed to have a beneficial effect on both clinical symptoms and in reducing the size of the stenotic lesions in these cases. Our patient remained asymptomatic clinically and improvement documented bronchoscopically was judged to be 80% during steroid therapy.

One other patient with bronchostenosis due to sarcoidosis has been reported with serial pulmonary function testing.⁵ The bronchostenoses in this patient were identified on bronchograms, but not identified bronchoscopically.

Benatar and Clark also noted that airway resistance can be increased in sarcoidosis secondary to lymph node compression, endobronchial granulomas, or narrowing and distortion of small airways due to fibrosis. Our patient initially demonstrated mild reduction in airways flow with volume restriction and compromised diffusing capacity. After initiation of therapy the vital capacity and diffusing capacity reverted to normal but decreased flow in the effort independent portions of the flow volume loop persisted. Based on bronchoscopic findings the obstruction appears to be in larger airways. Measurements of lung recoil were not performed, however an increase in the latter due to fibrosis might logically mask a portion of the expected flow reduction.

The presence of endobronchial granulomas with bronchostenosis and minimal parenchymal fibrosis in our case confirms that chest roentgenograms can not always be relied upon as an index of involvement. We would agree with recent statements by DeRemee⁶ that earlier aggressive treatment of sarcoidosis with adrenal corticosteroids is mandated irrespective of symptoms. The disparity between symptoms, pulmonary functions and endobronchial findings in this case emphasize the importance of this point.

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CONTRAINDICATIONS Hypersensitivity to oxycodone or acetaminophen.

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New Diagnostic Methods, V. A. Hosp., Hot Springs, SD, Feb. 2. 1 hr. AMA Category I credit. Contact: Off. of CME, USD School of Med., Sioux Falls, SD 57105.

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, International Hotel, New Orleans, LA, Feb. 2-4. 13 hrs. AMA Category I credits. Fee: \$215. Contact: International Med. Ed. Corp., 64 Inverness Drive, E., Englewood, CO 80112.

Management of Respiratory and Cardiac Failure, South Seas Plantation, Captiva Island, FL, Feb. 5-9. 22½ hrs. AMA Category I credits. Fee: \$185—ACCP members, \$200—non members. Contact: Dale E. Braddy, Dir. of Ed., Am. College of Chest Physicians, 911 Busse Highway, Park Ridge, IL 60068.

Biofeedback and Stress Reduction, Human Services Center Aud., Yankton, SD, Feb. 7. 3 hrs. AMA Category I credit. Contact: Bill Arbes, Ph.D., Dept. of Psychiatry, USD School of Med., Sioux Falls, SD 57105.

Coronary Disease, Exercise Testing, and Cardiac Rehabilitation, La Mansion Del Norte, San Antonio, TX, Feb. 9-11. 13 hrs. Category I credits. Fee: \$202. Contact: International Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

EKG Interpretation and Arrhythmia Management, Sheraton, Fort Lauderdale, FL, Feb. 9-11. 15 hrs. AMA Category I credits. Fee: \$202. Contact: International Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Genetic Factors in Mental Illness, V. A. Hosp., Fort Meade, SD, Feb. 15. 1 hr. AMA Category I credit. Contact: Off. of CME, USD School of Med., Sioux Falls, SD 57105.

Coronary Disease, Exercise Testing, and Cardiac Rehabilitation, Sahara Hotel, Las Vegas, NV, Feb. 23-24. 13 hrs. Category I credits. Fee: \$202. Contact: International Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Clinical Application of Biofeedback in Medical Practice, McKennan Hosp. Aud., Sioux Falls, SD, Feb. 27. No fee. 3 hrs. AMA Category I credits. Contact: Bill Arbes, Ph.D., Dept. of Psychiatry, 2501 W. 22nd St., Sioux Falls, SD 57101, (605) 339-6785.

Topics in Psychosomatic and Behavioral Medicine, U. of Michigan Medical Center, Ann Arbor, MI, Feb. 27-28. Cat. I credits. Contact: Office of CME, Towsley Center for CME, U. of Mich. Med. School, Ann Arbor, MI 48109.

International Conference on Occupational Lung Disease, Hyatt Regency-Embarcadero, San Francisco, CA, Feb. 27-March 2. Contact: Mr. Dale Braddy, Dir. of Ed., American College of Chest Physicians, 911 Busse Highway, Park Ridge, IL 60068.

Paramedic Parameters '79, Los Angeles Hilton, Los Angeles, CA, Feb. 28-March 2. Contact: U. of Southern Calif., Assoc. Dean, Postgraduate Div., 2025 Zonal Ave., Los Angeles, CA 90033.

March

Live Clinic—Multiple Sclerosis, V. A. Hosp., Fort Meade, SD, March 1. 1 hr. AMA Category I credit. Contact: Off. of CME, USD School of Med., Sioux Falls, SD 57105.

Second Annual Vail Cancer Conference Kiandra-Talisman Lodge, Vail, CO, March 3-10. Fee: \$220. 22 hrs. AMA Category I credits. Contact: Cancer Conference, P.O. Box 11366, Denver, CO 80211.

Ophthalmology Clinical Conference, U. of Iowa, Iowa City, IA, March 4. Category I AMA credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Medicine, Iowa City, IA 52242.

Physician Compensation and Contracting, Frenchman's Reef, St. Thomas, Virgin Islands, March 5-7. Fee: \$335. Contact: Registrar, Aspen Systems Corp., 20010 Century Blvd., Germantown, MD 20767.

18th National Conference on the Detection and Treatment of Breast Cancer, Atlanta, GA, March 5-8. Category I AMA credits. Contact: American College of Radiology, 6900 Wisconsin Ave., Chevy Chase, MD 20015.

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EKG Interpretation and Arrhythmia Management, International Hotel, New Orleans, LA, March 9-11. 15 hrs. AMA Category I credits. Fee: \$202. Contact: International Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, Americana of Bal Harbour, Miami, FL, March 9-11. 13 hrs. AMA Category I credits. Fee: \$215. Contact: International Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Fourth Annual Vail General Surgery Conference, The Mark, Vail, CO, March 10-17. Fee: \$220. 22 hrs. AMA Category I credits. Contact: General Surgery Conference, P.O. Box 11366, Denver, CO 80211.

Advances in Pediatrics, U. of Mich. Med. School, Ann Arbor, MI, March 14-16. Contact: Office of CME, U. of Michigan Med. School, Towsley Center for CME, Ann Arbor, MI 48109.

(continued)

Life Stages (Getting Past the Oedipus Complex), V. A. Hosp., Fort Meade, SD, March 15. 1 hr. AMA Category I credit. Contact: Off. of CME, USD School of Med., Sioux Falls, SD 57105.

Coronary Disease, Exercise Testing, and Cardiac Rehabilitation, Water Tower Hyatt, Chicago, IL, March 16-18. 13 hrs. Category I AMA credits. Fee: \$202. Contact: International Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

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First Annual Vail Gerontology Conference, Lion Square Lodge, Vail, CO, March 17-24. Fee: \$220. 22

hrs. AMA Category I credits. Contact: Gerontology Conference, P.O. Box 11366, Denver, CO 80211.

General Surgery, U. of Mich. Med. School, Ann Arbor, MI, March 22-23. Contact: Office of CME, U. of Michigan Med. School, Towsley Center for CME, Ann Arbor, MI 48109.

International Conference on Tuberculosis, Royal Plaza Hotel, Orlando, FL, March 22-24. 15 hrs. AMA Category I credits. Fee: \$160—ACCP members, \$175—non members. Contact: Dale E. Braddy, Dir. of Ed., Am. College of Chest Physicians, 911 Busse Highway, Park Ridge, IL 60068.

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Table of Contents: page 3

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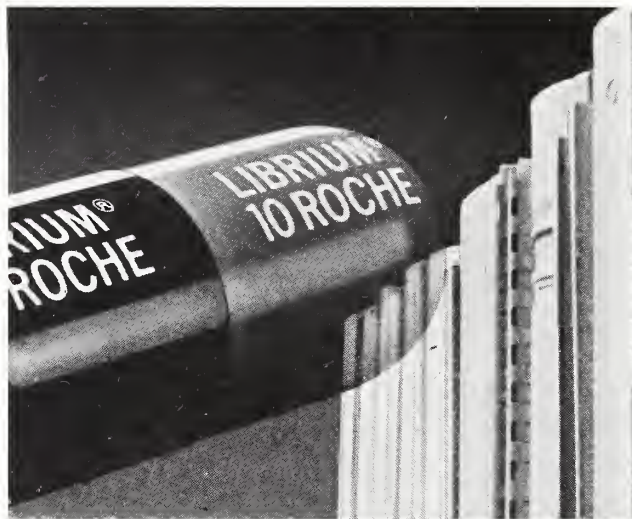
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SCIENTIFIC ARTICLES

- 7 Manic-Depressive Illness or Paranoid Schizophrenia?
Barton Adrian, M.D.
H. T. Hermann, M.D.
- 15 Ob-Gyn Lecture #12
Review of Breech Presentation
Curtis M. Adams, M.D.
- 25 Clinicopathological Conference
Forty-Eight Year Old Man with Edema of the Lower
Extremities and Hodgkin's Disease
F. S. Brady, M.D.
R. D. Willis, M.D.
John F. Barlow, M.D.

FEATURES

- 10 Laboratory Aids
South Dakota Pathology Page
- 18 This Is Your Medical Association
- 21 South Dakota AFP Chapter News
- 37 Future Meetings

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Manic-Depressive Illness or Paranoid Schizophrenia?

Barton Adrian, M.D.*

H. T. Hermann, M.D.**

Abstract

Rigorous clarification of the diagnostic distinctions between schizophrenia and manic-depressive illness has been defined by Abrams et al. This was applied to groups of patients diagnosed schizophrenic (paranoid or schizo-affective) and manic-depressive. A 20 percent error in the diagnosis of schizophrenia was found, a diagnosis of manic-depressive illness being more appropriate. Patients already having the diagnosis of manic-depressive illness had approximately a 40 percent chance of a previous diagnosis of schizophrenia.

The following paper presents a review and critique of the accuracy of diagnosis of all patients receiving the diagnosis of paranoid schizophrenia on two wards of a Veterans Administration Hospital. Of the 51 patients whose charts were reviewed, 33 had a diagnosis of schizophrenia; 19 of these were either paranoid type or schizo-affective. There has been recent interest in the need to clearly distinguish whether one is treating a patient who has paranoid schizophrenia or manic-depressive illness.¹

It appears certain now that Lithium has a real and practical advantage over other neuroleptic drugs in the treatment of manic-depressive disease. The failure to diagnose manic-depressive illness and the inclusion of such within the group of patients diagnosed paranoid schizophrenia, therefore, becomes potentially an error in inappropriate treatment of certain psychiatric conditions. Although manic disorders, when acute, yield better to sedative neuroleptics, like Chlorpromazine or Thioridazine, in the long run it seems that patients will do

better and relapse less frequently with the use of Lithium.

A study done by Abrams, Taylor and Gaztanaga² were reported, in which they found that of 41 patients receiving the diagnosis of paranoid schizophrenia in 247 consecutive admissions to an acute treatment psychiatric unit, only two of these 41 satisfied research criteria for schizophrenia, whereas half the 41 satisfied research criteria for mania. We were curious then to study a group of our patients with the diagnosis of paranoid schizophrenia, using similar criteria, in order to see whether we might also be dealing with a large group of unrecognized persons experiencing manic-depressive illness.

METHOD

(We adapted the following criteria from Abrams, Taylor and Gaztanaga):

- "1. For schizophrenia (a) through (d) required
 - (a) Formal thought disorder (blocking, non-sequiturs, neologisms, word approximations, verbigeration).
 - (b) Emotional blunting (restricted affective

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range and intensity with absence of emotional responsivity, warmth, finer ethical and moral notions, feelings of love for friends and family).

- (c) Incomplete verbal auditory hallucinations or autochthonous delusional ideas (excluding grandiose notions of wealth, high birth, or depressive delusions of sin, poverty, guilt).
 - (d) No evidence of coarse brain disease, medical illness known to produce psychiatric symptoms, past or present hallucinogenic drug ingestion, or psychostimulant drug abuse associated with the index admission.
2. For mania (a) through (d) required
- (a) Hyperactivity.
 - (b) Rapid/pressured speech.
 - (c) A euphoric, expansive, or irritable mood.
 - (d) No evidence of coarse brain disease, medical illnesses known to produce psychiatric symptoms, or psychostimulant drug abuse associated with the index admission."

The problem list was examined on each patient record on two wards of the hospital. These problem lists are cumulative and list all problems that the patient has had since problem lists were kept.

Any record that had a diagnosis of schizophrenia, paranoid, chronic or schizophrenia, schizo-affective, was selected for study.

Records used included all past records of admission in VA Hospitals and any transfer summaries or records from other hospitals that were in the patient's chart. The study was done retrospectively, utilizing admission histories and mental status exams, discharge summaries, nursing notes and progress notes.

The records were taken at face value, i.e. the patient was assumed to have delusions even if the chart stated only that "the patient was delusional" and did not record the delusions themselves; records also used expressions such as "patient had ideas of reference" or "showed loose association of thoughts when interpreting proverbs"; they rarely gave verbatim reports of what patients said.

RESULTS

Fifty-one record files of patients were reviewed on the two wards; 33 of these had a diagnosis of schizophrenia and 19 of these were paranoid or schizo-affective in type.

Study was then made whether of these 19 the criteria of Abrams, Taylor and Gaztanaga were fulfilled:

11 fulfilled criteria as schiz paranoid

2 more also fulfilled the criteria for mania (Case #7 and Case #14)

1 also for manic depressive (Case #15)

1 also for depression (Case #1)

Of the remaining four cases diagnosed schiz paranoid:

2 were disqualified because of hallucinogenic drug use also involved (Case #16 and Case #17)

1 was disqualified because of questionable OBS findings from encephalitis (Case #18)

1 was disqualified because of failure to clearly fulfill any of the criteria (Case #19)

Of the fifteen patients who would clearly satisfy the criteria for schizophrenia there are four who also qualify for affective disorder and hence might be in a manic-depressive diagnostic category. A diagnosis of manic-depressive disease had not been made in these cases. "Assuming" these patients did have manic-depressive illness, then it would appear the risk of error in diagnosis is 4/19, or approximately 20 percent in this study.

In order to approach the subject of correct diagnosis from a different angle, the medical records section was asked to provide the records of manic-depressive patients who had been discharged from the hospital. These were then subjected to the same criteria used before.

There were nine sets of records for patients currently diagnosed as manic depressive; all had data which met the criteria for this diagnosis. Three had previously been diagnosed schizo-affective schizophrenia and one had been diagnosed schizophrenia with various type listing: chronic undifferentiated, hebephrenic, residual, paranoid, as well as schizo-affective. So, of the 9 diagnosed manic-depressive, 4 had been confused with schizophrenia at some time in the past.

COMMENT

Our finding of a 20 percent error in this small series (4 of 19) compares with 93 percent (39 of 41) in the study of Abrams, Taylor and Gaztanaga, these figures representing the error of making a diagnosis of schizophrenia, paranoid, when the preferable diagnosis would be manic-depressive illness. This might mean that there is a wide variation in diagnostic habits or skills.

One problem in research which depends on review of records is that the data base will supply assumptions and opinions in abundance but not the description of how these are drawn, requiring a high degree of trust and allowing little independent evaluation.

Schizophrenia may be peculiarly difficult for agreement on diagnosis; the research standards used by Abrams, Taylor and Gaztanaga for paranoid schizophrenia provide an explicit framework which likely exceeds in rigor what is used by many less careful diagnosticians.

Manic depressive illness might seem less subject to confusion with schizophrenia, but the evidence of history and research show this is not an easy distinction.

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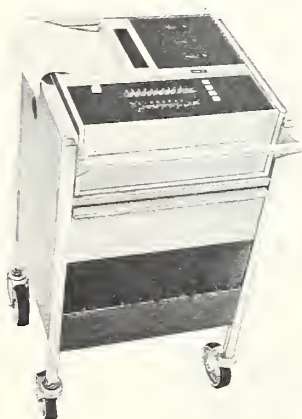
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Lecture #12

Review of Breech Presentation

Curtis M. Adams, M.D.*

INTRODUCTION

Nothing in obstetrics can be more frustrating for the scientific minded clinician than is breech presentation. Over the past 10 years, momentum has swung towards relying more heavily upon cesarean section.¹ This philosophy has been accepted by many, but conversely has been vehemently opposed on grounds of "losing the state of the art" by others. Which philosophy is correct, and which is incorrect? This article attempts to address this complex issue, based on literature review.

The frequency of breech presentation, excluding external cephalic version and multiple gestations is roughly 3 to 4%.² The type of breech at term is roughly frank 65 to 73% complete, incomplete 4.6 to 18.9%, and footling 12.5 to 28.5% respectively. These figures can change dramatically with decreasing weeks of gestation.⁶ Etiologic factors have been enumerated.³ In summary, one should suspect abnormality in the fetus, uterus, or pelvis when breech presentation arises. Despite diligent search, any factor can only be found in 6.8 to 8.0% of breech deliveries.

Perinatal mortality and morbidity are statistics which are commonly referred to in decisions on management. Corrected perinatal mortality was reviewed by Sinder and Wenstler.⁴ Their report summarized materials from 1945 to 1970 with a total perinatal mortality varying from 9.7 to 19.2%, with that of full sized infants 1.6 to 5.7%. Corrected perinatal mortality ranges from 0.4 to 3.5%.⁵ Also, the collected review from 1968 to 1972 reported

similar figures.⁵ One reason for these astronomical high figures is that small sized (602 to 1250 grams) premature infants in breech position occur statistically more frequently 30% vs. 10% ($p < 0.001$).⁵ This alone will not account for the measured perinatal death rate, as when 601 to 1250 grams and 1251 to 2500 are examined separately, the rate is approximately two times that of vaginal deliveries. When this group of patients was subjected to increased cesarean section rates, perinatal mortality improved ($p < 0.01$). Similar conclusions were reached by Goldenberg and Nelson,⁶ especially for breech infants weighing < 1500 gms. In autopsy evaluation of breech infants, Potter and Adair⁷ found death from traumatic hemorrhage in 42.4% of their cases; whereas, that for infants delivered by cesarean section was 6.5%. For full term infants (> 37 weeks or > 2500 gms.) the picture is roughly the same. Perinatal mortality ranges 3 to 4 times greater for vaginal delivery. By relying on increased cesarean sections, mortality can also be reduced ($0.01 < p < 0.05$).^{5,8} Mortality, however, is not the only consideration of the ill effects of breech delivery.

Morbidity in the form of brain trauma or asphyxia from prolapse of the umbilical cord, injuries to liver, spleen, adrenal, and spinal cord, fractures and dislocations is real. Injuries in some form have occurred in 7.0 percent of full term vaginal deliveries.⁵ When difficulties with partial extraction are encountered, the one minute Apgar score is low (0 to 6) in 40.6% of cases vs. 6.0% when no difficulties are encountered.⁵ Alexopolus notes that 7.2% of those injuries occurring are of a permanent and severe nature, in particular brain

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damage.⁹ Muller et. al. note that breech delivery is more apt to be followed by grades repeated, below normal arithmetic achievement, and recommendations for remedial measures than is cephalic delivery or cesarean section.¹⁰ Visual and auditive defects as well as speech disorder and emotional maladjustment have been noted to be increased by other observers.⁸

Prolapse of the umbilical cord is a serious complication resulting, if not treated promptly, in asphyxia and death. The frequency of cord prolapse in breech delivery is reported to be (3 to 5%).^{5,8,11,12} Factors thought to influence this figure are parity^{13,8,2} (multiparous>primiparous), type of breech (Footling>Frank),^{14,5,8} maternal age >30,¹³ artificial rupture of membranes, fetal age (>with increasing degree of prematurity),^{8,13} and dilatation of the cervix.¹⁵ Slate and Randal reported an equal incidence of prolapse at all degrees of cervical dilatation, except at full dilatation when the frequency increased dramatically. Perinatal mortality when prolapse occurs is 10 to 50%.^{11,2,5,8,13}

MANAGEMENT

Many solutions regarding the dilemma for the method of delivery of a breech infant have been expounded, however, tangible scientific approaches are elusive. More often one is likely to hear the statement, "I've delivered a lot of breeches;" or, "The last breech I delivered went smooth as silk." The following are many attempted solutions to this most complex issue.

One stand that has been used is to perform cesarean section on all primigravid patients. The thought of an untested pelvis has largely led to this chain of thought. It has also been shown that the one minute Apgar score is more often depressed in primigravidas.⁵ Of those patients followed by Alexopoulos for consequence of birth injury, or asphyxia, two-thirds were from primigravid mothers. Contrary to this, in review of (4,000) breech deliveries, it was found that difficulties in vaginal deliveries occurred equally frequently between primigravid and multiparous patients, 19.2 vs. 18.1% respectively.^{5,8} Perinatal mortality, likewise, seems to be distributed equally between the two groups, 3.8% vs. 3.0%.⁸ Indeed, when one considers the greater likelihood of umbilical cord prolapse in the multiparous group, this approach can be seriously questioned.

Neilson provided valuable work in reference to the large breech infant. This group (>8½ lbs.) represents 12.5% of the total breech population. When vaginal delivery was allowed, gross neurological signs occurred in both multiparous and primiparous patients in 7.1% and 24.3% respectively. Most im-

portantly, he noted that in the multiparous group, 86.5% had previously delivered an infant greater than 8 lbs. and, in the primiparous group, 53.3% were prolonged more than 42 weeks.¹⁶

The converse group which derives benefit from cesarean section is the premature less than 2500 gms. Soldberg et. al.⁶ point out the fact that one-third of all breeches are premature. With greater degree of prematurity, more likely was the likelihood of a presentation other than frank breech. Furthermore, with improvement in neonatal intensive care units, the long term prognosis for normal development is excellent.^{17,18}

A flat plate of the abdomen is a necessity in all contemplated vaginal deliveries to rule out hyperextension of the fetal head. Should the variant be found, cesarean section is the management of choice.^{19,20}

X-ray pelvimetry may be of some value. It has been shown that when clinical pelvimetry is compared to X-ray pelvimetry, there is a correlation in 2/3 of cases.¹ Also, when the conjugate vera is adequate (more than 12 cm.) and the pelvis considered absolutely normal, there is difficulty with a delivery in 17.0% of cases.⁵

The Zatuchni-Andros scoring index is an attempt at antepartum evaluation.²¹ It utilizes several factors: parity, gestational age, estimated fetal weight, previous breech, dilatation, and station. Bird,²² in a 6-year prospective study, concludes that patients with a score of 3 or less be submitted to immediate cesarean section, whereas those with a score of 4 or greater be allowed to labor. This, however, does not guarantee that the Apgar score will be >7 or that resuscitation efforts will not be required.

Piper forceps, prophylactically applied to the aftercoming head of breech infants, has been shown to be associated with a decrease in the perinatal mortality rate of infants between 1,000 and 3,000 grams ($p<0.01$).²³

The practice of external cephalic version has both proponents^{24,25,26,27} and opponents.²⁸ Results which can be anticipated are that the incidence of breech presentation will roughly be reduced by one-half (4.5 to 2.9%) in the most recent study.²⁷ Complications can be expected to occur even after placental localization with ultrasound, in approximately 5% of the cases.²⁷ These include uterine contractions, bleeding, rupture of membranes, and cord accident. Fetal death has also been reported,²⁹ presumably due to excess pressure resulting in placental abruption or cord accidents. Results are clearly influenced by gestational age, parity, amount of amniotic fluid, and experience of the clinician.

The opponents of operative delivery most often

adhere to the dictum of "once a cesarean, always a cesarean." Even this has come under scrutiny.^{30,31} In 276 patients with 423 deliveries after a section 57.5% were allowed a trial of labor, 70.4% delivered vaginally with no scar rupture. Weak scars are reported in 1.1 to 2.0% of the cases,^{30,31} with rupture occurring in 0.5%.³²

SUMMARY

In summary, an attempt has been made to draw upon previous publications for solutions to the multiple problems of management of breech presentations. It is clear that many cases mandate cesarean section: large fetus over 4,000 grams, hyperextension of the fetal head, Zatuchni-Andros index less than 4, footling presentation, and infants with a weight of less than 2,500 grams (although a lower limit must be determined by the related perinatal center). For the remaining cases other criteria such as pelvimetry (preferably X-ray), and ultrasound (which at best are "guesstimates")¹ must be employed if attempted vaginal delivery is entertained. Fetal malformation, uterine anomaly, and placental location must be searched for diligently. When finished, the fact remains that an unmolded fetal head must attempt passage through the bony pelvis, and possibly, undilated cervix. External cephalic version can reduce the incidence of breech presentation at term by approximately one-half, although the price paid is exposing everyone early in gestation to a 5.0% complication rate with the possibility of fetal death. Relying increasingly upon cesarean section dramatically reduces perinatal mortality and morbidity, while only slightly increasing maternal mortality with less severe morbidity. The correct cesarean section rate for breech presentation has yet to be determined.

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The Aberdeen District Medical Society elected **A. J. Janusz, M.D.**, president for 1979. Other officers include **John Christopher, M.D.**, vice president and **B. G. Welge, M.D.**, secretary-treasurer.

* * * *

Joseph Kass, M.D., Rosholt, has been named a Diplomat of the American Board of Family Practice. **A. J. Tieszen, M.D.** and **R. J. Zakahi, M.D.**, Pierre, have been notified of their recertification by the ABFP.

* * * *

Charles Pelton, M.D., Aberdeen, and **Keith W. Sehnert, M.D.**, Excelsior, Minnesota, were guest speakers at the "Health Promotion" seminar held at Presentation College, Aberdeen for health care administrators, RN's and LPN's.

* * * *

The Sioux Falls District Medical Society elected the following officers for 1979: **Lowell Hyland, M.D.**, president; **Dennis Johnson, M.D.**, vice president; **John Ochsner, M.D.**, secretary; and **Ronald Wyatt, M.D.**, treasurer.

* * * *

Stephen Haas, M.D., Rapid City, discussed exercise and the diabetic, how exercise helps control diabetes and medication and diet for the diabetic at the Black Hills Diabetes Association meeting held in Rapid City.

James Monfore, M.D., and **James DeGeest, M.D.** were presented with commemorative plaques for their ten and twenty years of service to the community of Miller during the Christmas party for the Hand County Memorial Hospital. Making the presentation representing the State Medical Association was **Bob Johnson**, Executive Secretary.

* * * *

Officers for 1979 for the Huron District Medical Society include **Arlan Zastrow, M.D.**, president; **Guillermo Huet, M.D.**, vice president, and **E. A. Hofer, M.D.**, secretary-treasurer.

Jorge E. Sanmartin, M.D., Rapid City, has been elected to fellowship in the American Heart Association Council of Clinical Cardiology.

* * * *

E. H. Heinrichs, M.D., Vermillion, participated in a panel discussion on child abuse televised by the PBS station in Brookings. This program was held immediately following the documentary "Raised in Anger", which was televised nationally.

* * * *

Christmas Seals announced that **Thomas Mead, M.D.**, Spearfish, was one of five South Dakotans receiving a tuition scholarship to attend the 4th Annual Super-course on respiratory disease sponsored by the American Lung Association of Louisiana and the American Thoracic Society of Louisiana. The course was held in New Orleans this past December.

* * * *

Lawrence F. Nelson, M.D., Webster, was re-elected president of the Whetstone Valley District Medical Society, and **Eldon Bell, M.D.**, Webster, was elected secretary.

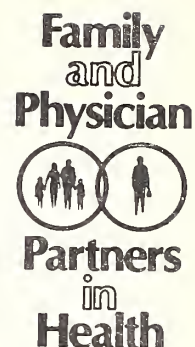
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Anthony Talbert, M.D., has opened his office in Rapid City for the practice of pediatrics and adolescent medicine. He previously practiced in Yankton.

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SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS
3001 South Holly Avenue
Sioux Falls, SD 57105



Results of Annual Survey of Family Practice Residency Programs Revised Nov. 15, 1978

I. Programs:

A. Total Approval Programs	348
B. Total Operating Programs (2 approved but not operating)	346
Community Hospital Based	60
University Based	58
University Affiliated or Administered	211
Military Hospital Based	17

II. Residents:

A. Total Residents	6,033
1. Total First Year Residents	2,318
2. Total Second Year Residents	1,986
3. Total Third Year Residents	1,729
B. Total Approved First-Year Positions	2,411
C. First Year Fill Rate	96.1%
D. Increase/Decrease Class Size by Year	

	1976-77	1977-78	1978-79
Class of '79	1,861	1,830	1,729
Class of '80	—	2,043	1,986
Class of '81	—	—	2,318

III. Residency Graduates:

A. Total July, 1978 residency graduates	1,548
B. Total graduates from family practice residency programs since January 1, 1970	4,942

SOC

The 1979 State Officers Conference of AAFP will be held in Kansas City, April 27-29. State officers are expected to attend and other members may attend. The annual Infectious Diseases Symposium will again be held that Friday, April 27, and preceded by a Family Practice Update, sponsored by Kansas University School of Medicine. Watch for mailings regarding these meetings.

SDSMA Meeting

Mark your calendar for the 1979 SDSMA meeting in Rapid City, June 8-10. We will have our annual SDAFP "nooner" on Saturday, June 9, at 12:15 p.m. Program to include "Potpourri from the SDAFP." Watch for program mailings.

THE ALUMNI ASSOCIATION

of the

SOUTH DAKOTA SCHOOL OF MEDICINE

has been established by the South Dakota Medical School Endowment Association. Among other activities, the Alumni Association serves as a source of information for graduates and assists in the organization of class reunions.

As of 1977 the South Dakota School of Medicine is a four-year degree granting school, and through the Alumni Association the school, and past and present students will be better served.

Contributions may be sent to:

**Alumni Association
South Dakota Medical School
Endowment Association
608 West Avenue, North
Sioux Falls, South Dakota 57104**

Forty-Eight Year Old Man with Edema of the Lower Extremities and Hodgkin's Disease

F. S. Brady, M.D.*

R. D. Willix, M.D., FACS**

Discussers

John F. Barlow, M.D., FCAP***

Editor

Case No. 757775

This 48-year-old male was admitted to Sioux Valley Hospital because of edema of the lower extremities of one week's duration. Two years prior to admission the patient had had swelling in the right neck and a mediastinal mass. Biopsy of the neck mass revealed nodular sclerosing Hodgkin's disease. He underwent a staging laparotomy with disease found in a small area of the spleen as well as in abdominal lymph nodes. He was felt to have Stage 3A disease and underwent a 9 month course of COPP (cyclophosphamide, vincristine, prednisone, procarbazine) therapy. The patient had extensive x-ray therapy to the neck and chest (estimated at over 4,000 radiation total dose). The patient had done well since that time. A few months prior to admission he had had some low grade fever and cough productive of yellowish sputum. He had some mild chest and back pain which was sharp and occurred in no localized area. The pain lasted for 6 weeks but disappeared. For several weeks prior to this admission, he had noticed recurrent low grade fever, particularly in the evenings and some cough with recurrence of the non-descript localized chest pain. One week prior to admission, he noted ankle, penile, and scrotal swelling and also noticed that his pants seemed to be getting quite tight. He also noticed some right upper quadrant aching and had shortness of breath on exertion but no shortness of breath on assuming the recumbent position. Examination in another hospital stated that he had prominent neck veins, distant heart sounds, a large liver as well as edema of the penis, scrotum, and lower extremities. His electrocardiogram showed ST elevation in leads II, III, and AVF. A Swan-Ganz catheterization was performed. Right atrial pressure

was 8 mm., right ventricular diastolic pressure was 8 mm., and pulmonary artery pressure was 8 mm. The patient was transferred.

PHYSICAL EXAMINATION: Height 5'11"; weight 176 pounds; pulse 110 per minute and regular; respirations 24 per minute and regular, blood pressure 110 systolic and 70 diastolic. Patient was alert and fully oriented lying in bed. Temperature 100°F. Also there was 6-8 mm. of paroxysm in the blood pressure with the left arm at a 45° angle.

Examination of the head and neck revealed no palpable lymphadenopathy. Thyroid was not enlarged. The chest was clear to auscultation and percussion. The jugular veins were distended 6 cm. above the right clavicle and jugular venous pulsations were visible at this level. The jugular venous pulse did not fall with inspiration and may even have increased slightly. There was marked hepatjugular reflux. The heart sounds were normal but somewhat diminished; however, no gallop sounds or murmurs were heard. There was a soft pericardial friction rub noted in systole and diastole, heard best at the left lower sternal border. The rub varied markedly with respiration being audible during inspiration. There was no palpable point of maximal intensity.

Examination of the abdomen revealed some tenderness in the right upper quadrant and the liver was palpable 5-6 cm. below the costal margin with a span of 15 cm. but no nodules were felt. No other palpable organs or masses were felt. There was 1+ pitting edema in the pretibial region in the ankles with edema of the scrotum and penis.

LABORATORY DATA: Urinalysis was hazy yellow, specific gravity 1.012, pH 7.0, negative for protein, glucose, ketone bodies, and hemoglobin; sediment one white cell per high power field, negative for red cells and casts. Hemoglobin 10.8 gm./dl; red count 3.16 million/mm³; hct, 31 vol/dl, mean corpuscular hemoglobin 32 micrograms, mean corpuscular volume 97 cubic micra, mean corpuscular hemoglobin concentration 32%. Total leukocyte count 8,700/mm³ with 60% segmented neutrophils, 2% neutrophilic bands, 4% eosinophils, 23% lymphocytes, and 2% monocytes. There was slight anisocytosis of the red cells and the platelets were normal in number and

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***Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Professor of Pathology, School of Medicine, University of South Dakota.

morphology. A zetacrit was 70% (markedly elevated). Fluorescent antinuclear antibody test was less than 1 to 10 dilution, pH was 7.40 pCO₂ 41 torr. CO₂ content 24mm/L, PO₂ 79 torr, oxygen saturation 91%. Lactic dehydrogenase was 288 IU/L (normal up to 270 IU/L), alkaline phosphatase 165 IU/L (normal up to 115 IU/L). Transaminase, total bilirubin, calcium, total protein, inorganic phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, and cholesterol were within normal limits. The intermediate strength PPD showed no induration. Six blood cultures and a urine culture revealed no growth. Cultures

were done because the temperature rose intermittently to 101°F after admission. A sputum culture revealed enterobacter aerogenes resistant to ampicillin and cephalothin and sensitive to carbenicillin, genatmicin, kanamycin, and tetracycline. Electrocardiogram was normal manifesting T-wave changes suggesting diffuse anterior septal wall ischemia, possible submyocardial infarction and clockwise rotation of the heart with low voltage QRS in the limb leads due to possible pericarditis. An echocardiogram showed changes consistent with pericardial effusion. A surgical procedure was performed.

CARDIAC CATHETERIZATION DATA:

	Normal	Before 340 cc of pericardial fluid withdrawn	After fluid withdrawn
right atrial pressure	5(mean)	—	12
right ventricular pressure (systolic)	30	32	34
right ventricular pressure (diastolic)	7	16	14
pulmonary artery pressure (systolic)	30	28	28
pulmonary artery pressure (diastolic)	10	15	14
pulmonary artery pressure (mean)	20	23	-
pulmonary wedge pressure	12	15	12
stroke volume	-	58.18 ml	-
stroke volume index	45 ± 15	29.09cc/m ²	-
cardiac output	-	6.4	-
cardiac index	2.5 ± 14m	3.2	-

DR. BRADY: In summary, this is a 40-year-old man who presents two years after a diagnosis of stage IIIA nodular sclerosing Hodgkin's disease which was treated with 9 months of COPP therapy and extensive radiation to the neck and chest in the range of 4,000 rads total dose. He had recurrent low grade fever, cough, non-descript, poorly localized chest pain, shortness of breath on exertion, edema, ascites, distended neck veins, Kussmaul's sign, pulsus paradoxus, tachypnea, tachycardia, distant heart sounds, pericardial friction rub, hepatomegaly with tenderness, and no palpable point of maximum intensity.

Abnormal laboratory findings included a normochromic, normocytic anemia of mild degree, markedly elevated zetacrit, elevated lactic dehydrogenase (LDH), and alkaline phosphatase. He had a positive sputum culture for enterobacter as well as low voltage and ischemia suggested on electrocardiogram. There were bilateral pleural effusions. Right ventricular diastolic pressure, pulmonary artery diastolic pressure and pulmonary wedge pressure were essentially equal. After 340 cc. of fluid was removed from the pericardium; these pressures, while lower, still remained elevated and essentially

equal. Stroke volume index was low but because of tachycardia, the cardiac index was normal.

It is obvious this patient had pericardial disease and I believe the other abnormalities were due to the resulting congestive process. Because of the lack of orthopnea, it is doubtful he had primary myocardial disease. He did have pericardial effusion as demonstrated by pericardiocentesis but he also had a constrictive component because the catheterization pressures, after the fluid was removed, remained elevated and equal, and because right ventricular diastolic pressure was greater than one-third the right ventricular systolic pressure. I believe he had effusive constrictive pericarditis and that a pericardiectomy was performed.

I would like to review the pericardium and its diseases before elaborating on the differential diagnosis in this patient. Normal pericardium is smooth, transparent, and contains 25 to 35 ml of serous fluid with the same composition as lymph. The proposed functions of the pericardium include providing a frictionless surface for contractions, restraining against cardiac overdistention, supporting the heart in a stable position, and guarding against infection from the outside.

Besides inflammation, any disease with generalized edema could cause increased fluid in the pericardial sac. Symptoms of pericardial effusion and/or pericarditis include exertional dyspnea, abdominal swelling, peripheral edema, palpitations, cough, vague chest pain, and fever. Physical findings include raised jugular venous pressures and pulsations, distant and muffled heart sounds, hepatomegaly, ascites, pulsus paradoxus, peripheral edema, pleural effusions, and cardiomegaly.

Pulsus paradoxus is a greater than normal inspiratory decline in arterial pressure. It is usually present if the fall in pressure is greater than 6 to 8 mm. of mercury with quiet breathing. This sign may also occur with hyperventilation, respiratory obstruction, asthma, congestive failure with dyspnea and shock accompanied by air hunger. However, usually the decline with these entities is smaller than with pericardial effusion. There are three explanations for the decreased left ventricular stroke volume during pericardial effusion with tamponade. These are as follows: (1) with high intrapericardial pressure during the respiratory cycle, venous pressure in the pulmonary veins outside the pericardium falls more than the atrial pressure during inspiration resulting in pulmonary venous pooling and decreased cardiac filling, (2) a tense pericardium is further stretched during inspiration by movement of the diaphragm and sternum and this further compresses the heart and reduces left ventricular filling, (3) in a taut pericardium, persistence and augmentation of the normal inspiratory increase in systemic venous return expands the right heart volume and increases intraepicardial pressure. This is followed by an increase in the left heart volume and pressures several beats later during expiration. If tamponade is severe enough, just the filling of the right heart decreases filling of the left heart.

In addition to the above findings, an elevated erythrocyte sedimentation rate or zeta crit, low voltage QRS complexes on electrocardiogram, atrial fibrillation (with constrictive disease), T wave flattening or inversion (depending on how long the condition has existed), identical right and left ventricular diastolic pressures and identical rise in the right atrial and pulmonary wedge pressures occur. The right ventricular diastolic pressure should be greater than one third of the right ventricular systolic pressure.

Acute pericarditis can be limited to the pericardium or be part of a systemic disease. Causes might be of infectious, collagen disease, metabolic, chemical or neoplastic origin. Myocardial infarction, trauma, and irradiation all can produce pericarditis or the disease may be idiopathic. Chronic constrictive

pericarditis has essentially the same origins. The typical progression of inflammation in the pericardium includes stages of pain, effusion, organization, resorption, and constriction. The degrees of each stage may vary and they can merge into a subacute effusive constrictive type necessitating pericardiectomy.

Studies from Vanderbilt¹¹, India^{13,16}, and other centers showed evidence that the constrictive condition is a fairly common result of effusions of most etiologies. However, no percentages were given. A study from Ohio State of 12 patients presenting only with chest pain, fatigue, and dyspnea accompanied by completely normal studies, found that they could demonstrate occult constrictive pericarditis with cardiac catheterization studies immediately after infusing 1 liter of warm saline over 6 to 8 minutes. None of the controls at catheterization showed changes with the volume expansion. Of the 12 patients mentioned above, 7 had a previous history of pericarditis. All 12 had an excellent response to surgery. It was felt that these patients were possibly in a spectrum passing toward frank constrictive pericarditis. Surgery was felt to be warranted if the patients were symptomatic and catheterization abnormalities were demonstrated after rapid volume expansion.

There is a multitude of causes for pericarditis as I have mentioned but it would seem practical to limit the differential diagnosis in this case to three—*infectious*, *neoplastic*, (particularly Hodgkin's disease of the pericardium), and *irradiation*. The normal white count, negative blood cultures, and low grade fever are points against an infectious etiology. The presence of enterobacter in sputum cultures requires, however, that infection must be considered.

Hodgkin's disease is a disease of the lymphatic system and it would rarely involve the pericardium except by contiguous spread. In my sources on the disease I found only one reported case of Hodgkin's disease causing pericarditis. In addition there are no signs of active disease on chest x-ray and the patient has had intensive therapy. All of the above make Hodgkin's disease an unlikely cause of the pericardial process in this case.

Therefore, the radiation received would be the best choice as the causative factor. It is only in recent years that radiation has been included as a factor in causing significant pericarditis. However contemporary studies single it out as a probable cause of not only pericarditis, but myocardial infarction, myocarditis, aortitis, and valvulitis. Pericarditis is most frequently seen after irradiation therapy for Hodgkin's disease, breast, esophageal

or lung carcinoma. In Hodgkin's disease, the incidence of pericarditis can be as high as 25 to 30%. Most symptomatic patients present within one year of therapy which has totaled at least 4,000 rads. The spectrum of the disease can vary from transient or chronic asymptomatic effusion, progression to pericardial tamponade due to effusion or fibrous constrictive pericarditis. In Baltimore a study followed 81 patients for two years after treatment by 4,000 rads for Hodgkin's disease. Within two years, 24 patients or 30% met x-ray criteria for the presence of pericardial fluid. These criteria involved an increase in 1.5 cm. over baseline transverse cardiac diameter or a distinct change in cardiac configuration without such an increase. Of the 24 patients 14 had transient asymptomatic effusions, 10 had persistent effusion, and 5 required partial pericardiectomy for signs and symptoms of pericardial tamponade. Six of the 10 with pericardial effusions had low voltage patterns in electrocardiogram and 5 out of those 6 underwent pericardiectomy. Echocardiograms indicated effusion in all 10 who had pericardial effusion by x-ray and demonstrated transient effusion in two other cases. Examination of the fluid showed a high protein content with a predominance of lymphocytes. The pericardium examined from those who underwent surgery had mild to moderately thickened parietal pericardium over the anterior surface with a mild reaction of the visceral layer over the right atrium and ventricle, but the left ventricle and its overlying pericardium were not involved with much inflammatory reaction. Also no recurrent Hodgkin's disease was evident grossly or histologically in the excised pericardium. The operated patients remained asymptomatic with normal cardiac diameters on x-ray and normal voltage on electrocardiograms. It was felt that the role of pericardiocentesis was limited to patients with pericardial tamponade or pre-operatively because of risk of circulatory collapse during induction when right heart filling pressures decreased due to vasodilatation. Except for microbiologic cultures, diagnostic tests are limited in pericarditis.

The natural history of effusions is variable but some believe that even in transient effusion, there is a good chance that cardiac constriction may develop later. The indications for surgery include all symptomatic patients and those asymptomatic patients with a 5 cm. increase in cardiac diameter persisting 6 to 9 months.

Another reason for surgery rather than simply tapping the fluid is to rule out recurrent Hodgkin's disease, which is difficult if not impossible to diagnose on a fluid specimen.

Followup of asymptomatic patients with increased

transverse cardiac diameter includes clinical evaluation, chest x-ray, electrocardiograms, and echocardiograms. If symptoms develop, right heart catheterization should be performed to check for pericardial tamponade. If the pressures are normal, the patients can be followed further. If pressures are increased or left ventricular volume or functions are impaired, surgery is recommended. Except in emergency situations, the patient is prepared for surgery by admission to the hospital for digitalization and stabilization of fluid and electrolyte status. Antibiotics are started as indicated for infectious causes. Surgery can be augmented with the use of cardiopulmonary bypass.^{1,7} This shortens the operative time, decreases the risk of cardiac injury and blood loss and facilitates dissection by permitting work on a fibrillating heart allowing more complete removal of the visceral pericardium. Surgical results have been good to excellent in 85 to 90% of the patients. With the risk of sudden death from pericardial tamponade, increasing evidence suggesting that most effusions will eventually result in pericardial constriction and an operative mortality of just 5%, pericardiectomy has been increasingly popular as therapy for refractory pericardial effusion as well as pericardial tamponade and chronic pericardial constriction.

Dr. Brady's Diagnosis

Effusive-Constrictive Pericarditis Due to Irradiation.

DR. WILLIX: How do you know that this patient did not have just acute pericarditis and pericardial effusion and did indeed have pericardial constriction?

DR. BRADY: The long history would suggest other than acute pericarditis and he had signs of constrictive disease also on physical exam and by cardiac catheterization especially after pericardial fluid was removed. Equalization of pressures in the right heart would also suggest pericardial constriction.

DR. WILLIX: This patient did have some confusing elements in his clinical picture. The presence of fever made us suspect that he might have an acute pericarditis. The other possible explanation for the fever, of course, was recurrent Hodgkin's disease. That latter diagnosis was further supported by anorexia and malaise which this man exhibited. It should be pointed out, however, that our diagnosis was much easier since the patient was referred to this hospital from Yankton, South Dakota, with a diagnosis of pericardial constriction which had been made quickly after admission by the passage of a Swan-Ganz catheter demonstrating the equalization of pressures in the right atrium, right ventricle,

pulmonary artery and capillary wedge pressures in diastole. Therefore, the diagnosis of constrictive pericarditis had already been made before the patient reached this hospital. This is extremely helpful, of course, in patient evaluation.

I would like to make several points about the surgical approach to pericardial disease. In patients with pericardial effusion, the treatment of choice is pericardiocentesis. If the pericardial effusion recurs, a tube can be left in the pericardium for drainage. If this does not succeed, a small operation can be performed through a left anterior thoracotomy called a pericardial window where a small segment of pericardium is removed and the pericardial fluid can drain into the pleural cavity. This operation relieves pressure on the heart and allows for periodic thoracentesis, rather than having to perform periodic pericardiocenteses. However, if only that procedure is done in a patient such as the one discussed today, the patient's symptoms would not have been relieved since he had constrictive as well as effusive disease. This was proved when we removed pericardial fluid during catheterization and the patient's pressures continued to be elevated as described by Dr. Brady.

The surgical approach that we take in cases of constrictive disease such as this is to use a midline sternotomy incision opening the parietal pericardium and then stripping the visceral pericardium from the heart. The difficulty of this task depends on the stage of the disease. In this particular patient, the visceral pericardium could be removed from the entire heart including both ventricles and both atria and superior and inferior vena cava without the need for cardiopulmonary bypass. The cardiopulmonary bypass pump is always in the room in case the heart is entered during the pericardial stripping. This is a reasonably common complication of this type of operation. If this occurs, we can put the patient on the pump, protect the heart with cardioplegic solution and continue to strip the pericardium on the fibrillating heart. The stripping of the pericardium can be extremely difficult and may have to be removed in centimeter segments. It should be stressed that if you only remove the parietal pericardium, you do not relieve the constriction. Constriction is present because of the fibrotic inflammatory process in the visceral pericardium. In this patient the parietal pericardium was perhaps a cm. in thickness and the visceral pericardium perhaps 0.5 cm. in thickness.

Another complication in addition to entering the heart particularly in the right ventricle and right atrium is an injury to a coronary artery. You cannot visualize the coronary arteries at all. You have

to rely fairly heavily on the knowledge of the normal positioning of the coronary vessels. Recurrence of pericardial constriction may recur or persist if the entire visceral pericardium is not removed.

This man's admission weight was 176 pounds. During the initial cardiac catheterization he lost 4 to 5 pounds from fluid withdrawn. His ascites disappeared prior to surgery and after surgery his weight dropped to 157 pounds. On visits to the office, the patient reports that he is back working and has had no recurrence of his pericardial edema or ascites. His weight is stable at 155 pounds.

I think we must consider postradiation pericarditis with pericardial effusion and constriction as a more common disease than has formerly been thought. We all see many patients who present with bilateral pleural effusions several years after having undergone a radical mastectomy for carcinoma of the breast. Some of these patients may not have recurrent carcinoma. It is not uncommon to do a thoracentesis on such patients and the fluid is reported to be free of malignant cells. The patient continues to have a progressive downhill course and we assume that the patient has recurrent carcinoma. In this group of cases it is very possible that we are missing cases of effusive-constrictive pericarditis. It should be pointed out, in the presence of pleural effusion, pericardial effusions are much more difficult to detect by echocardiogram. The diagnostic potential of the echocardiogram can be optimized if the patient has bilateral pleural taps before the echocardiogram study is performed.

It is important to remember in patients who have pericardial constriction or pericardial effusions accompanied by systemic hypotension that the low blood pressure results from low stroke volume subsequent to decrease atrial and left ventricular filling during diastole because of the constriction or pericardial effusion. In this situation, in which the pericardial effusion is impairing the function of the left ventricle in this manner, it is appropriate to load the patient with fluid in spite of the fact that the patient has distended neck veins. If pericardial disease is the problem, load them with fluid before you get them to a referral center. This will, at least, insure that the heart will eject the maximal stroke volume from the left ventricle possible under the conditions of the pericardial disease. Administering fluid would certainly seem to be the wrong thing to do since the patient has distended neck veins but it is of value and will help to temporize the situation until the pericardial fluid or constriction can be relieved.

One point worth noting is that I think one should always remember that myxedema can cause peri-

cardial effusion, low voltage changes on the electrocardiogram, and hypotension.

The cardiovascular surgeon likes to see patients with pericardial disease early. For instance, it has been shown in a patient with tuberculous pericarditis, the treatment of choice is drug therapy followed by elective pericardiectomy in order to circumvent a more difficult operation for more advanced constrictive pericarditis which is the outcome of almost all patients who develop tuberculous pericarditis.

One of these studies that Dr. Brady eluded to was at Ohio State.¹² It suggested early pericardiectomy for all patients with pericarditis and effusion. A liter of saline was given as a fluid load in patients who had normal right ventricular, right atrial, and right pulmonary artery diastolic pressures and pulmonary wedge pressures. In a normal patient there will be no equalization of these pressures. However, provided this infusion is done in the catheterization laboratory over a 6-8 minute period of time, patients who have what they call subacute pericarditis will show equalization of these pressures. These patients are then selected for elective pericardiectomy to prevent the development of constrictive pericarditis.

In closing I would like to again emphasize that constrictive pericarditis is more common than we realize. Certainly patients who have received radiation for breast cancer or Hodgkin's disease or patients who have received radiation for other chest neoplasms must be considered to have pericardial disease if they enter the hospital with symptoms of hypotension, anorexia, and pleural effusions. It is a reasonably simple procedure to insert a Swan-Ganz catheter as a diagnostic test and make a diagnosis of pericardial constriction on that basis.

DR. BRADY: Would you follow your patients who have been irradiated for Hodgkin's disease as has been suggested with a chest x-ray every month for the first six months and every two months for the next year and then yearly?

DR. WILLIX: I would go one step further. I would obtain a baseline echocardiogram and then probably repeat that test yearly. I would also do a chest x-ray at least every six months the first few years and then yearly.

*DR. SULLIVAN: Can you differentiate pericardial effusion from pericardial constriction?

DR. WILLIX: The echocardiogram is usually only going to demonstrate the presence of fluid, but it can tell you if the pericardium is extremely thick. However, the diagnostic test for pericardial constric-

tion is a cardiac catheterization. The method for doing this is to place a needle into the pericardial sac and measure the diastolic pressures in the right atrium, right ventricle, pulmonary artery as well as pulmonary wedge pressure. The diastolic pressures in these cardiac cavities should be similar to the pericardium. The pericardial fluid is then evacuated and the pressure in the pericardium is again measured. This lowers the pericardial pressure and should restore right atrial, ventricular, pulmonary artery and wedge pressures to normal if only pericardial fluid is producing the constriction. However, the pulmonary artery, right ventricle and right atrial diastolic pressures remained equal in this case. This indicates that there is continuing constriction in spite of the absence of pericardial fluid.

Therefore, you need to measure both intrapericardial and intracavity cardiac pressures for best diagnostic performance. The real problem arises in the patient who has progressed from effusive-constrictive pericarditis to total constriction. The pericardial fluid is lost and pericardial cavity obliterated. You cannot measure pericardial pressures. In this situation, your only course is just to measure the intracavity cardiac pressures.

DR. BARLOW: I will show some slides of this case of effusive-constrictive pericarditis. A mixture of fibrous tissue, hemorrhage, and fibrin are seen. There are scattered large irregular nuclei in fibroblasts as one might expect from the radiation process but this is not striking. The findings are typical of what have been described by the Fagardo.⁵ (Fig. 1)

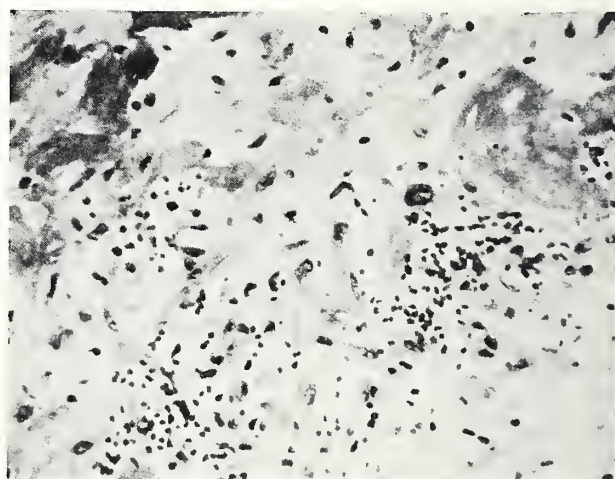


Figure 1
Fibrin, inflammatory cells and an occasional large fibroblast are seen in the irradiated thickened pericardium.

I feel one must make a few basic distinctions in dealing with pericardial disease. Normally 20 to 50 cc.'s may be found in the pericardial sac. Increased pericardial fluid or hydropericardium may

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be seen in congestive heart failure, nephrosis, or myxedema. Whether or not interference with diastolic filling of the ventricle by this pericardial fluid occurs depends on the amount of fluid, the rate of accumulation of fluid, underlying cardiac disease, and nature of the fluid. This interference with ventricle filling is called pericardial tamponade.

A serous, fibrinous, serofibrinous or serosanguineous effusion may occur with a variety of diseases including rheumatic fever, uremia, collagen disease (lupus), virus (coxsackie), radiation, myocardial infarction, neoplasm, or post-myocardial infarction syndrome. All of these may or may not be accompanied by enough fluid accumulated fast enough to produce pericardial tamponade. A usual but not invariable sequence of events is slow resorption of fluid to occur along with progressive fibrosis of the pericardial layers with the eventual onset of pericardial constriction. In the interval as fluid is being absorbed and fibrosis of pericardium is occurring, a state of effusive-constrictive pericardial disease results. Eventually there is little or no fluid present and a marked fibrous process involving both the parietal and visceral pericardium is present. As it has been pointed out, it is the visceral pericardial fibrosis that most commonly produces interference with diastolic filling of the ventricles in the end stage of constrictive pericarditis. It is obvious, therefore, as Dr. Willix has pointed out, that diagnosis early in the course of the disease before dense scarring has taken place would make removal of the fibrous material easier. The cause of constrictive pericarditis includes many causes mentioned above but tuberculous, histoplasmic and purulent pericarditis as well as radiation and rheumatoid arthritis head the list of causes of constrictive pericarditis.

FINAL ANATOMIC DIAGNOSIS: EFFUSIVE CONSTRICTIVE PERICARDITIS DUE TO RADIATION

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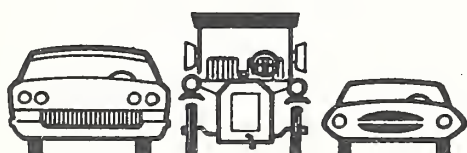
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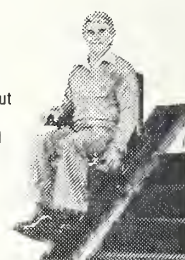
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Future Meetings

February

Dermatology, Mayo Foundation Outreach Seminar, McKennan Hospital Aud., Sioux Falls, SD, Feb. 23-24. AMA Cat. I & AAFP prescribed credits. Contact: Sec., Med. Ed., McKennan Hospital, 800 E. 21st St., Sioux Falls, SD 57101.

Practical Approaches to Depression, Family Health Center, Aberdeen, SD, Feb. 24. No fee. 2 hrs. AMA Category I credits. Contact: Bill Arbes, Ph.D., Dept. of Psychiatry, 2501 W. 22nd St., Sioux Falls, SD, 57101, (605) 339-6785.

Antimicrobial Susceptibility Testing, Lab. of Clinical Medicine, down stairs classroom, Sioux Falls, SD, Feb. 24. 3 hrs. AMA Category I credits. Contact: Richard A. Jaqua, M.D., LCM, 1212 S. Euclid Ave., Sioux Falls, SD 57105 339-1212.

Clinical Application of Biofeedback in Medical Practice, McKennan Hosp. Aud., Sioux Falls, SD, Feb. 27. No fee. 3 hrs. AMA Category I credits. Contact: Bill Arbes, Ph.D., Dept. of Psychiatry, 2501 W. 22nd St., Sioux Falls, SD 57101, (605) 339-6785.

March

Live Clinic—Multiple Sclerosis, V. A. Hosp., Fort Meade, SD, March 1. 1 hr. AMA Category I credit. Contact: Off. of CME, USD School of Med., Sioux Falls, SD 57105.

Allergy and Clinical Immunology, Mayo Memorial Aud., U. of Minn., Minneapolis, MN, March 1-3. Fee: \$150. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455.

Hospice-Developmental and Operational Issues, Marriott Inn, Bloomington, MN, March 2-3. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455.

Second Annual Vail Cancer Conference Kiandra-Talisman Lodge, Vail, CO, March 3-10. Fee: \$220. 22 hrs. AMA Category I credits. Contact: Cancer Conference, P.O. Box 11366, Denver, CO 80211.

Ophthalmology Clinical Conference, U. of Iowa, Iowa City, IA, March 4. Category I AMA credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Medicine, Iowa City, IA 52242.

Invitational Conference on Psychiatric Factors of Drug Abuse, Spring Hill Center, Wayzata, MN, March 4-6. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455.

Cardiology Today, U. of Iowa, Iowa City, IA, March 5-8. Fee: \$275. 30 hrs. AAFP & AMA credits. Contact: Attn: C. W. White, M.D., Cardiovascular Div., U. of IA., Iowa City, IA 52242.

Ophthalmology Clinical Conference, U. of Iowa, Iowa City, IA, March 7. AMA Category I credits. Contact: Office of CME, 285 Med Labs, U. of Iowa, Iowa City, IA 52242.

Fourth Annual Vail General Surgery Conference, The Mark, Vail, CO, March 10-17. Fee: \$220. 22 hrs. AMA Category I credits. Contact: General Surgery Conference, P.O. Box 11366, Denver, CO 80211.

Medical Records—Planning or Documentation, V. A. Hosp., Fort Meade, SD, March 15. 1 hr. AMA Category I credit. Contact: Off. of CME, USD School of Med., Sioux Falls, SD 57105.

Psychiatry for the Primary Care Physician, St. Paul Ramsey Med. Center, St. Paul, MN, March 15-16. Fee: \$100. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455.

Fourth Annual Vail Internal Medicine Conference, The Mark, Vail, CO, March 17-24. Fee: \$220. 22 hrs. AMA Category I credits. Contact: Internal Medicine Conference, P.O. Box 11366, Denver, CO 80211.

First Annual Vail Pediatrics Conference, Lion Square Lodge, Vail, CO, March 17-24. Fee: \$220. 22 hrs. AMA Category I credits. Contact: Pediatrics Conference, P.O. Box 11366, Denver, CO 80211.

Hematology, Mayo Foundation Outreach Seminar, McKennan Hospital Auditorium, Sioux Falls, SD, March 23-24. Category I AMA & AAFP credits. Contact: Dept. of Med. Ed., McKennan Hospital, 800 E. 21st St., Sioux Falls, SD 57101.

Sexual Attitude Reassessment Seminar, U. of Minn., Minneapolis, MN, March 23-24. Fee: \$90. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455.

Psychopharmacological Treatment of the Medically Ill, Huron Reg. Med. Center, Huron, SD, March 27. 2 hrs. AMA Category I credit. Contact: Bill Arbes, Ph.D., Dept. of Psychiatry, USD School of Med., Sioux Falls, SD 57105.

Hypertension Update—1979, L'hotel de France, Bloomington, MN, March 28. Fee: \$10. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455.

State of the Art Review of Cardio-pulmonary Diseases, Coffman Union Theater, U. of Minn., Minneapolis, MN, March 29-31. Fee: \$125. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455.

Identifying and Handling Stress in Yourself and Others, McKennan Hosp. Aud., Sioux Falls, SD, March 30-31. Contact: Bill Arbes, Ph.D., Dept. of Psychiatry, USD School of Med., Sioux Falls, SD 57105.

April

Intensive Course in Pediatric Nutrition, U. of Iowa, Iowa City, IA, April 2-6. AMA Category I credits. Contact: Office of CME, 285 Med Labs, U. of Iowa, Iowa City, IA 52242.

American Cancer Society, National Conference, Urologic Cancer-1979, Los Angeles Hilton Hotel, Los Angeles, CA, April 4-6. 15½ hrs. AMA Category I credits. Contact: Urologic Cancer Conference, American Cancer Society, 777 Third Ave., New York, NY 10017.

(continued)

Brain Stem Syndromes, V. A. Hospital, Fort Meade, SD, April 5. 1 hr. AMA Category I credit. Contact: Office of CME, USD School of Med., 2501 W. 22nd St., Sioux Falls, SD 57105 339-7573.

Iowa Perinatal Meeting, Des Moines, IA, April 5-6. AMA Category I and AAFP credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Multiple Sclerosis—Update, V. A. Hospital, Hot Springs, SD, April 6. 1 hr. AMA Category I credit. Contact: Office of CME, USD School of Med., 2501 W. 22nd St., Sioux Falls, SD 57105 339-7573.

Coronary Disease, Exercise Testing, and Cardiac Rehabilitation, Hyatt Regency, Atlanta, GA, April 6-8. 13 hrs. Category I AMA credits. Fee: \$202. Contact: International Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Family Practice Review, U. of Mich. Med. School, Ann Arbor, MI, April 16-20. Contact: Office of CME, U. of Michigan Med. School, Towsley Center for CME, Ann Arbor, MI 48109.

Social Disaster and Mental Illness, V. A. Hospital, Fort Meade, SD, April 19. 1 hr. AMA Category I credit. Contact: Office of CME, USD School of Med., 2501 W. 22nd St., Sioux Falls, SD 57105 339-7573.

Otolaryngology Clinical Conference, U. of Iowa, Iowa City, IA April 20. AMA Category I and AAFP credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Nephrology, Mayo Foundation Outreach Seminar, McKennan Hospital Auditorium, Sioux Falls, SD, April 20-21, Category I AMA & AAFP credits. Contact: Dept. of Med. Ed., McKennan Hospital, 800 E. 21st St., Sioux Falls, SD 57101.

EKG Interpretation and Arrhythmia Management, Sahara Hotel, Las Vegas, NV, April 20-22. 15 hrs. AMA Category I credits. Fee: \$202. Contact: International Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Radiation Therapy Seminar, U. of Iowa, Iowa City, IA, April 26. AMA Category I and AAFP credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, Marriott Hotel, Dallas, TX, April 27-29. 13 hrs. AMA Category I credits. Fee: \$215. Contact: International Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

EKG Interpretation and Arrhythmia Management, Quality Inn, Hilton Head, SC, April 27-29. 15 hrs. AMA Category I credits. Fee: \$202. Contact: International Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Management of Common Problems in Office Practice—Update, The Fairmont, New Orleans, LA, April 27-May 1. Contact: New Orleans Graduate Medical Assembly, 1430 Tulane Ave., New Orleans, LA 70112.

1979 American Occupational Health Conference, Disneyland Hotel, Anaheim, CA, April 30-May 4. Fee: \$45—members, \$65—non members. Contact: American Occupational Med. Assoc., Box P, 150 N. Wacker Dr., Chicago, IL 60606.

May

Seventh Annual Surgical Intensive Care Symposium, May 4-7. Eden Roc Hotel, Miami Beach, FL. 20 hrs. AMA Category I credits. Contact: Div. of CME, D23-3, U. of Miami School of Medicine, P.O. Box 016960, Miami, FL 33101.

Ninth Annual Meeting of the Great Plains Organization for Perinatal Health Care, Radisson South Hotel, Bloomington, MN, May 10-12. Contact: Mrs. Virginia Rittenour, Coord., Great Plains Organization, Box 50, 420 Delaware St., S.E., Minneapolis, MN 55455.

Cardiology Today, U. of Iowa, Iowa City, IA, May 14-17. AMA Category I and AAFP credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Physician Compensation and Contracting, The St. Francis, Union Square, San Francisco, CA, May 17-19. Fee: \$335. Contact: Registrar, Aspen Systems Corp., 20010 Century Blvd., Germantown, MD 20767.

Advances in Internal Medicine, Towsley Center for CME, U. of Michigan Med. School, Ann Arbor, MI, May 21-25. Contact: Office of CME, U. of Michigan Med. School, Towsley Center for CME, Ann Arbor, MI 48109.

Gastric Bypass, U. of Iowa, Iowa City, IA, May 24-25. AMA Category I and AAFP credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

June

South Dakota State Medical Association Annual Meeting, Howard Johnson Motor Lodge, Rapid City, SD, June 8-10. Contact: SDSMA, 608 West Ave., North, Sioux Falls, SD 57104.

Coronary Disease, Exercise Testing, and Cardiac Rehabilitation, Inn on the Park, Toronto, Canada, June 8-10. 13 hrs. AMA Category I and AAFP credits. Fee: \$202. Contact: International Med. Ed. Corp., 64 Inverness Dr., East, Englewood, CO 80112.

Intensive Course in Pediatric Nutrition, U. of Iowa, Iowa City, IA, June 11-15. AMA Category I and AAFP credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Fourteenth Annual Meeting of the Rocky Mountain Neurosurgical Society, Jackson Lake Lodge, Jackson Hole, WY, June 13-17. Contact: Ralph J. Kaplan, M.D., Sec., U. of Oklahoma Health Sciences Center, P. O. Box 25606, Oklahoma City, OK 73125.

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, Lincolnwood Hyatt, Chicago, IL, June 15-17. 13 hrs. AMA Category I and AAFP credits. Fee: \$215. Contact: International Med. Ed. Corp., 64 Inverness Dr., East, Englewood, CO 80112.

10th International Congress for Suicide Prevention and Crisis Intervention, Ottawa, Ontario, Canada, June 17-20. Fee: \$125. Contact: Secretariat, IASP Congress, Suite 700, 71 Bank St., Ontario, Canada K1P 5N2.

Scientific Seminar on Advances in Pediatrics, Sylvan Lake, Custer, SD, June 20-22. AMA Category I and AAFP credits. Contact: Thomas Aceto, Jr., M.D., Dept. of Pediatrics, U. of S.D. School of Med., McKennan Hosp., Sioux Falls, SD 57101.

Northern Michigan Summer Program, Shanty Creek Lodge, Bellaire, MI, June 25-29. Contact: Office of CME, U. of Michigan Med. School, Towsley Center for CME, Ann Arbor, MI 48109.

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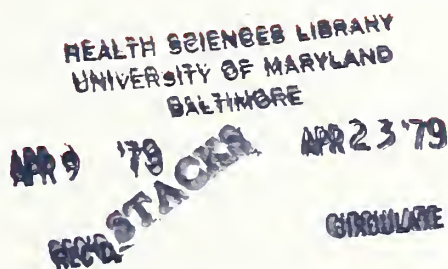


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SCIENTIFIC ARTICLES

- 9** Review of Sympathomimetic Bronchodilators
 Brian L. Kaatz, Pharm.D.

- 21** Clinicopathological Conference
 Thirty-Seven Year Old Man with History of
 Chronic Ulcerative Colitis and Recent
 Onset of Jaundice
 Robert Seidel, M.D.
 James R. Reynolds, M.D.
 John F. Barlow, M.D.

FEATURES

- 15** South Dakota AFP Chapter News

- 31** Council Meeting Highlights

- 32** Laboratory Aids
 South Dakota Pathology Page

- 35** Doctor Jeckel and Doctor Greed
 Winston B. Odland, M.D.

- 41** Future Meetings

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CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. **Drug Dependence:** Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychological dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. **Use in Pregnancy:** Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. **Use in Children:** Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: **Cardiovascular:** Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. **Central Nervous System:** Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. **Gastrointestinal:** Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. **Allergic:** Urticaria, rash, ecchymosis, erythema. **Endocrine:** Impotence, changes in libido, gynecomastia, menstrual upset. **Hematopoietic System:** Bone marrow depression, agranulocytosis, leukopenia. **Miscellaneous:** A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in the evening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in the morning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

MERRELL-NATIONAL LABORATORIES Inc.

Cayey, Puerto Rico 00633

Direct Medical Inquiries to:

MERRELL-NATIONAL LABORATORIES

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References: 1. Citations available on request—Medical Research Department, MERRELL RESEARCH CENTER, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon, R.H., and Leyland, H.M.: A Comprehensive Review of Diethylpropion Hydrochloride. International Symposium on Central Mechanisms of Anorectic Drugs, Florence, Italy, Jan. 20-21, 1977.

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8-3921 (YS67A)

Review of Sympathomimetic Bronchodilators

Brian L. Kaatz, Pharm.D.*

Chemical manipulation of drugs active on the autonomic nervous system has resulted in relatively specific drug activity for a number of diseases. Beta adrenergic drugs currently play an important role in the pharmacotherapy of asthma and other reversible obstructive lung diseases. Modification of the prototype drugs epinephrine and isoproterenol has produced a separation of cardiac stimulatory effects from bronchodilation. The use of drugs with primarily bronchial activity and minimal cardiac effects is advantageous for the treatment of chronic obstructive lung disease. This discussion reviews the newer, more selective agents including metaproterenol and terbutaline, and compares them with the traditionally used adrenergic bronchodilators.

PHARMACOLOGY OF BRONCHODILATORS

Because of the diverse actions of the autonomic nervous system it has been divided into the adrenergic and cholinergic systems.¹ This classification is based on the chemical transmitters released at the junctions of the effector organs. In 1948, the adrenergic system was subdivided into alpha and beta receptors to distinguish important differences in their physiologic effects.² In 1967, Lands further noted differences among the beta-adrenergic receptors and suggested still another subdivision.³ From these studies it was shown that cardiac muscle and bronchial smooth muscle actions are controlled by different receptors, namely beta-1 (cardiac), and beta-2 (bronchial dilation). See Table I.

Experimental models have demonstrated the important role of an intracellular mediator, cyclic AMP (cyclic adenosine 3', 5' monophosphate), in

asthma. A reduction in intracellular cAMP increases bronchial muscle tone.⁴ In contrast, an increase of intracellular cAMP relaxes bronchial smooth muscle. This proposed relationship of cAMP and muscle tone is the basis for the mechanism of action of commonly used drugs in asthma and related diseases.

Table I

ADRENERGIC SYSTEM EFFECTS

Alpha

- bronchial smooth muscle contraction
- skin and visceral smooth muscle constriction

Beta 1

- increased heart rate
- increased contractile force
- lipolysis
- intestinal relaxation

Beta 2

- bronchodilation
- vasodilation
- glycogenolysis
- uterine relaxation

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The beta adrenergic stimulants act by stimulating adenyl cyclase (at A) to produce more cAMP while theophylline and its derivatives (aminophylline) exert their bronchodilatory action by inhibiting the enzyme phosphodiesterase (B) thereby also resulting in increased levels of cAMP. See Figure 1. By virtue of their differing sites of action the beta adrenergic drugs and the theophylline type drugs can exert a potentiating bronchodilatory effect in the asthmatic patient. Lower doses of each can be used in combination to reduce unpleasant side effects.¹⁸

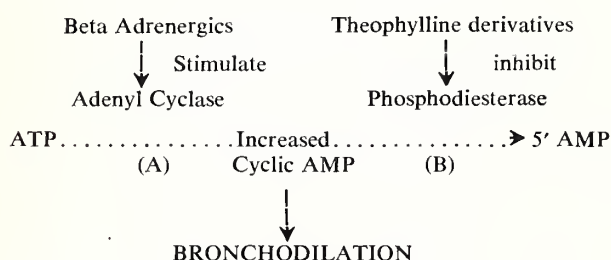


Figure 1

EPINEPHRINE

Epinephrine has potent effects on both alpha and beta adrenergic receptors. It is best utilized in acute asthmatic episodes, but is excluded from chronic regimens due to its intense stimulation of the heart and peripheral blood vessels. The drug has a very short duration of action due to its rapid inactivation by catechol-o-methyl transferase (COMT) and monoamine oxidase (MAO), which primarily occurs in the liver. Epinephrine is not effective orally and must be either inhaled or administered by the subcutaneous, intramuscular, or (rarely in asthma) intravenous routes. Side effects are common and include palpitations, severe headaches, tachycardia, increased blood pressure, tremors, anxiety, and weakness. Ventricular arrhythmias may occur with epinephrine especially in patients with preexisting organic heart disease. The dose for acute asthmatic attacks in adults is 0.1 to 0.5 ml of 1:1000 aqueous epinephrine injected subcutaneously. This dose may be repeated as needed but not more than every 20 to 30 minutes.

EPHEDRINE

Ephedrine has been used as an oral agent in the treatment of asthma.⁵ The mode of action is similar to that of epinephrine because it also stimulates both alpha and beta receptors. There are, however, several pharmacologic differences. Ephedrine has seven to ten times longer duration of action, is active orally, is more stimulating to the central nervous system

and is less potent than epinephrine. The smooth muscle dilation from ephedrine is less than that of epinephrine and is of practical value alone only in mild asthma. Although ephedrine is often included in combination products there is recent evidence that this inclusion may result in increased risk of toxicity without an increase in bronchodilation.⁶ Common side effects include increased heart rate and blood pressure, dizziness, restlessness, insomnia, and irritability. Tolerance to bronchodilation seems to develop quickly. Effectiveness may return when medication schedule is interrupted for 3 or 4 days.¹⁹ The usual dose of ephedrine varies from 25-50 mg given four to six times daily, with side effects often being the limited factor. Its usefulness alone is minimal with the advent of more effective agents.¹⁹

ISOPROTERENOL

Isoproterenol is the prototype compound for the newer sympathomimetic amines. It is generally classified as a pure beta stimulant and is therefore more specific for the treatment of bronchospastic diseases. The activity of isoproterenol, however, does not distinguish between beta-1 and beta-2 receptors and thus may result in unwanted cardiac side effects. The drug relaxes bronchial and gastrointestinal smooth muscle, lowers peripheral vascular resistance and has a positive inotropic and chronotropic action on heart muscle. Isoproterenol dilates previously constricted pulmonary vessels which can exaggerate the ventilation-perfusion inequality. This can increase hypoxemia despite effective bronchodilation. Acute toxicity is generally less than that of epinephrine but arrhythmias and death have occurred. Side effects are due primarily to beta-1 stimulation and include palpitations, trembling, headaches, weakness, dizziness, and tachycardia. Activity of isoproterenol is short lived because of rapid metabolism by COMT and MAO. Isoproterenol is most often used as an injection and aerosol.

The aerosol should be used no more than every three to four hours because excessive use can lead to decreased effectiveness or in some cases an exacerbation of bronchospasm.⁷ Because of its short duration of action and frequency of side effects (up to 30%), the newer more specific beta-2 adrenergic agents offer a more desirable choice.

ISOETHARINE

Isoetharine (Bronkosol, Bronkometer) is another compound that exhibits pure beta activity and is more specific for beta-2 receptors. It is available in the United States only as an aerosol and a nebulizing solution. Advantages over isoproterenol include few-

er side effects and perhaps a slightly longer duration of action though it is probably not greatly superior.⁷ Available as a nebulizing solution, it is quite valuable and widely used.

Table II			
Drug	Beta Adrenergic Activity		Duration
	Cardiac (B-1)	Bronchial (B-2)	
epinephrine	4	4	1
ephedrine	4	3	2
isoproterenol	3	4	3
isoetharine	2	4	3
metaproterenol	2	4	4
terbutaline	1	4	5

Key: Beta adrenergic activity
1=least activity 4=most activity
Duration
1=shortest duration 5=longest duration

METAPROTERENOL

Metaproterenol (Metaprel, Alupent) is a chemically related compound which has a longer duration of action than isoproterenol. It has greater beta-2 specificity and thus has less cardiovascular effects.⁸ Both of these properties can be explained by the modified chemical structure which prevents metabolism by COMT.

Numerous studies have compared metaproterenol with other adrenergic agonists. In a two month trial Chervinsky and Belinkoff compared the aerosol forms of metaproterenol and isoproterenol.⁹ Metaproterenol demonstrated a statistically significant increase in one-second force expiratory volume (FEV₁) at one hour. Other parameters also were increased. Aerosolized metaproterenol had a four hour duration of effective action while isoproterenol had a duration of less than 60 minutes.¹⁰ Maximum improvement in FEV₁ was 41% with metaproterenol and 22% with isoproterenol. Adverse reactions were infrequent in both studies.

In a study of oral sympathomimetic amines metaproterenol exhibited a longer duration of action than isoproterenol. Palpitations, trembling, and dizziness occurred with each drug.¹¹ Pharmaceutical company literature states approximately 8% of patients using the metaproterenol inhaler and 17% of patients taking tablets experience some adverse effect. These may include tachycardia, nervousness, tremor, and palpitations. With an increase in dose the observed beta-1 activity becomes more apparent and results in increased cardiovascular side effects. In humans an average of 40% of an oral dose is ab-

sorbed. It is metabolized by the liver and excreted in the urine. Metaproterenol is available as oral tablets, syrup, and aerosol. The usual adult dose is 20 mg three or four times daily. The aerosolized form can be administered as frequently as every three or four hours, but should not exceed 12 inhalations in one day.

TERBUTALINE

Terbutaline (Bricanyl, Brethine) has recently become available in the United States. The drug is very similar to metaproterenol in chemical structure but a slight modification renders it more beta-2 specific. When administered by the parenteral routes both terbutaline and metaproterenol demonstrate diminished beta-2 specificity. This may be due to higher blood levels achieved by these routes.⁷ Oral and subcutaneous terbutaline has been compared with epinephrine, metaproterenol, and ephedrine. Pulmonary function tests have shown that 5 mg of oral terbutaline is at least equivalent in bronchodilation to 20 mg metaproterenol and has a longer duration of action. When compared with oral ephedrine, terbutaline had a more rapid onset, longer duration, and greater activity.¹²

Other studies have also demonstrated a superiority of terbutaline over epinephrine, metaproterenol, and isoproterenol.¹³⁻¹⁶ Terbutaline given subcutaneously in a dose of 0.25 mg produced a similar response in ventilatory capacity, onset and duration to that of 0.25 mg epinephrine. The 0.5 mg dose of terbutaline provided an even greater improvement in airflow without a clinically significant increase in adverse effects.¹⁴ In subjects with chronic obstructive airway disease, 0.5 mg of subcutaneous terbutaline showed greater increases in FEV₁ and forced vital capacity than 0.25 mg epinephrine.¹⁵ There were no serious side effects. Similar findings were observed in acute asthmatic children.¹⁶

Terbutaline aerosol has been favorably compared with aerosols of isoproterenol and metaproterenol.¹⁷ A trial comparing 1.5 mg inhaled metaproterenol with 0.5 mg terbutaline showed the latter to have a significant increase in FEV₁ and peak expiratory flow rate (PEFR) after five hours. Cardiac rate and blood pressure remained unchanged with both drugs.

Terbutaline is available as an injectable solution and as oral tablets. There are plans for marketing an aerosolized product in the future. Side effects of oral terbutaline include skeletal muscle tremor in up to 20% of patients. This is a dose-related effect which is a direct stimulation of beta-2 receptors located in skeletal muscle. This "physiological" tremor can be aggravated by too much coffee or other stimulant

medications or doses taken too frequently. No muscle tremor has been reported following inhalation.¹⁷ Other side effects include palpitations, dizziness, nervousness, and tinnitus, although these are fairly rare and tend to decrease in severity and frequency with continued administration. The usual dose is 5 mg every six to eight hours. A dose of 2.5 mg three times daily is recommended for children 12 to 15 years of age and for patients who cannot tolerate the adverse reactions. The dosage of the aerosol is one or two inhalations every six to eight hours with a two minute interval between the first and second inhalations.

SUMMARY

Many drugs and therapeutic measures are important in the therapy of asthma and chronic obstructive pulmonary disease. These include theophylline and derivatives, sympathomimetic amines, cromolyn sodium, and oral and inhaled corticosteroids. The role of the sympathomimetic amines is best determined on an individual patient basis. All of these drugs should be used with caution in individuals with hypertension, coronary artery disease, congestive heart failure, hyperthyroidism, diabetes, cardiac arrhythmias, or when there is a sensitivity shown. A relative loss of beta-2 specificity may be observed upon parenteral therapy. This might be due to increased blood levels achieved as compared with inhalation therapy thereby directly stimulating the cardiovascular and central nervous systems. Thus aerosolized administration may be a valuable method of administering the beta-2 adrenergic medications. From data generated in clinical trials, terbutaline or metaproterenol would seem to be the adrenergic bronchodilators of choice for both short and long term therapy. These more specific drugs may replace older and less effective medications.

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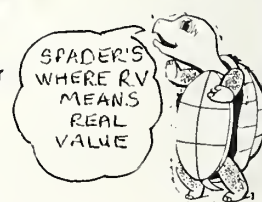


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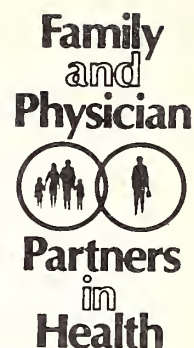
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AAFP Resident Activities Fact Sheet

Produced materials to assist family practice residents with documentation of experiences. Materials are available upon request to AAFP headquarters . . .

Published booklet "The Choice is Yours: Family Practice in Rural and Inner-City Areas," to provide information on advantages and problems associated with rural and inner-city family practice . . .

Achieved success with Joint Commission on Accreditation of Hospitals in gaining revisions in its "Accreditation Manual," which will permit departments of family practice to be organized and operated in the same manner as other departments . . .

Revised and updated the Academy's booklet on "Family Practice in Hospitals," which outlines methods of obtaining privileges and setting up a Department of Family Practice . . .

Strives continually to make the U.S. Congress aware of the effect utilization review requirements will have on small hospitals with limited resources . . .

Provides documentation to Congress and HEW that family practice residency graduates are setting up practice in areas of need, thus helping to assure a continued level of federal fiscal support . . .

Continues the RAP program, a confidential evaluation program for residencies to assure the continued availability of high quality education . . .

Appoints voting resident representatives to national Academy commissions and committees . . .

Appoints voting delegates to Academy's Congress of Delegates . . .

Supports development of state resident organizations within the Academy framework . . .

Continues to sponsor National Conference of Family Practice Residents for resident members to increase their involvement in Academy activities . . .

Expanded from quarterly to bimonthly "AAFP Resident and Student Newsletter" in the monthly AAFP Reporter . . .

Continues efforts with FDA in the area of patient information and patient package inserts . . .

Conducted Mental Health Workshops for the Impaired Physician and surveyed family practice residencies to determine nature and extent of physician impairment and whether or not preventive programs exist in the residencies . . .

Conducts Workshops on Practice Management . . .

Waived registration fee for residents attending AAFP Annual Scientific Assembly . . .

Continues to offer Academy-sponsored disability, life and retirement plans at competitive premiums to resident affiliate members of AAFP.

AAFP Student Activities Fact Sheet

Expanded Hotline (which assists senior medical students unmatched by National Intern and Resident Matching Program) to year-round basis to facilitate students finding available residency positions . . .

Began preparation of a uniform directory, listing pertinent information about each family practice residency program (directory to be available for distribution in May 1979) . . .

Produced complimentary informational packets giving helpful hints on how to establish family practice special interest groups . . .

Provided funding for speakers to address meetings of family practice special interest groups . . .

Appoints voting medical student representatives to national key Academy commissions and committees . . .

Continues to sponsor National Conference of Student Affiliate Member Representatives to increase student involvement in AAFP activities . . .

Created Bylaws provisions to provide mechanism for AAFP state chapters to charter student affiliate component chapters . . .

Expanded from quarterly to bimonthly the "AAFP Resident and Student Newsletter" in the monthly AAFP Reporter . . .

Continues liaison and participated in national meetings of Student National Medical Association and American Medical Student Association . . .

Sponsors workshops to brief practicing physicians on requirements and availability of part-time teaching positions in residency programs . . .

Through extensive testimony by Academy officers and strong support at the chapter level, was instrumental in efforts to have signed into law, the Health Professions Educational Assistance Act. The bill provided \$39 million for the support of family practice residency programs in fiscal year 1977 and \$45 million in 1978, \$45 million in 1979 and \$50 million in 1980 . . .

Assisted in development of family practice residencies to reach an all-time high of approximately 2,500 first-year positions . . .

Waived registration fee for medical students attending AAFP Annual Scientific Assembly . . .

Provides student lounge for informal conferences during AAFP Annual Scientific Assembly . . .

Provides reception for students during AAFP Annual Scientific Assembly . . .

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While the Medical Necessity Program is the responsibility of the Blue Shield Association, these specialty groups working in their respective areas of practice have voluntarily assisted in the programs, by helping identify procedures which are, in most circumstances, of dubious current usefulness.

The following is a list of those procedures already identified:

Bronchoscopy—with injection of contrast medium for bronchography
Bronchoscopy—with injection of radioactive substance
Ligation of internal mammary arteries, unilateral
Ligation of internal mammary arteries, bilateral
Radical hemorrhoidectomy, Whitehead type, including removal of entire pile bearing area
Omentopexy for establishing collateral circulation in portal obstruction
Kidney decapsulation, unilateral
Kidney decapsulation, bilateral
Perirenal insufflation
Nephropexy: fixation or suspension of kidney (independent procedure), unilateral
Circumcision, female
Hysterotomy, non-obstetrical, vaginal
Supracervical hysterectomy: subtotal hysterectomy, with or without tubes and/or ovaries, one or both
Uterine suspension
Uterine suspension, with presacral sympathectomy
Ligation of thyroid arteries (independent procedure)
Hypogastric or presacral neurectomy (independent procedure)
Angiocardiography, single plane, supervision and interpretation in conjunction with cineradiography
Angiocardiography, multi-plane, supervision and interpretation in conjunction with cineradiography
Angiocardiography, utilizing CO₂ method, supervision and interpretation only
Angiography—coronary, unilateral selective injection supervision and interpretation only, single view unless emergency
Angiography—extremity, unilateral, supervision and interpretation only, single view unless emergency
Protein bound iodine (PBI)
Icterus index

Basal metabolic rate (BMR)

Phonocardiogram with interpretation and report, and with indirect carotid artery tracing or similar study

Ballistocardiogram

Fabric wrapping of abdominal aneurysm

Extra-intra cranial arterial bypass for stroke

Fascia lata by stripper for lower back pain

Fascia lata by incision and area exposure, with removal of sheet for lower back pain

Ligation of femoral vein, unilateral or bilateral for post-phlebitic syndrome

Excision of carotid body tumor, with or without excision of carotid artery for asthma

Sympathectomy, thoracolumbar, unilateral or bilateral for hypertension

Sympathectomy, lumbar, unilateral or bilateral for hypertension

Splanchnicectomy, unilateral or bilateral for hypertension

We do not recommend that physicians categorically discontinue these procedures. Almost every procedure can be medically justified in a specific instance. We do recommend, however, that each physician determine whether the results of any procedure justify the cost.

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PERCOCET-5® ^{Tablets} II

Brief Summary of Prescribing Information

DESCRIPTION Each tablet of PERCOCET®-5 contains 5 mg oxycodone hydrochloride (WARNING: May be habit forming), 325 mg acetaminophen (APAP).

INDICATIONS For the relief of moderate to moderately severe pain.

CONTRAINDICATIONS Hypersensitivity to oxycodone or acetaminophen.

WARNINGS **Drug Dependence** Oxycodone can produce drug dependence of the morphine type and, therefore, has the potential for being abused. Psychic dependence, physical dependence and tolerance may develop upon repeated administration of PERCOCET®-5, and it should be prescribed and administered with the same degree of caution appropriate to the use of other oral narcotic-containing medications. Like other narcotic-containing medications, PERCOCET®-5 is subject to the Federal Controlled Substances Act.

Usage in ambulatory patients Oxycodone may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. The patient using PERCOCET®-5 should be cautioned accordingly.

Interaction with other central nervous system depressants Patients receiving other narcotic analgesics, general anesthetics, phenothiazines, other tranquilizers, sedative-hypnotics or other CNS depressants (including alcohol) concomitantly with PERCOCET®-5 may exhibit an additive CNS depression. When such combined therapy is contemplated, the dose of one or both agents should be reduced.

Usage in pregnancy Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. Therefore, PERCOCET®-5 should not be used in pregnant women unless, in the judgment of the physician, the potential benefits outweigh the possible hazards.

Usage in children PERCOCET®-5 should not be administered to children.

PRECAUTIONS **Head injury and increased intracranial pressure** The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a pre-existing increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.

Acute abdominal conditions The administration of PERCOCET®-5 or other narcotics may obscure the diagnosis or clinical course in patients with acute abdominal conditions.

Special risk patients PERCOCET®-5 should be given with caution to certain patients such as the elderly or debilitated, and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, and prostatic hypertrophy or urethral stricture.

ADVERSE REACTIONS The most frequently observed adverse reactions include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients, and some of these adverse reactions may be alleviated if the patient lies down.

Other adverse reactions include euphoria, dysphoria, constipation, skin rash and pruritus.

DOSAGE AND ADMINISTRATION Dosage should be adjusted according to the severity of the pain and the response of the patient. It may occasionally be necessary to exceed the usual dosage recommended below in cases of more severe pain or in those patients who have become tolerant to the analgesic effect of narcotics. PERCOCET®-5 is given orally. The usual adult dose is one tablet every 6 hours as needed for pain.

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EDO-603E379

Thirty-Seven Year Old Man with History of Chronic Ulcerative Colitis and Recent Onset of Jaundice

Robert Seidel, M.D.*

James R. Reynolds, M.D.**

Discussers

John F. Barlow, M.D.***

Editor

Case No. 771 802

This 37 year old caucasian male was admitted to Sioux Valley Hospital with painless jaundice of 4 to 5 days duration.

Nine days prior to admission, the patient noted churning pain in the right upper quadrant and the epigastrium after attending a picnic. The pain lasted several hours and subsided. The patient was able to work hard for the next 4 or 5 days but did note some clay-colored stools and dark urine. When he went home, his wife noted icteric sclerae two days prior to admission. He had also had some itching in the hands and antecubital fossae, but his appetite remained good. There was no nausea or vomiting. The patient had five to six loose stools for four days prior to admission. The patient had had known ulcerative colitis since age 16 but had few exacerbations, the latest being seven to eight years prior to admission. He was able to hold a job as an executive and his health had otherwise been excellent.

Five years prior to admission, the patient had an episode of abdominal pain and had an exploratory laparotomy at which time a liver biopsy showed very mild nonspecific triaditis. Liver function tests at that time were within normal limits. The patient had a negative review of systems other than the previous appendectomy.

PHYSICAL EXAMINATION: Temperature 98.4°F., pulse 80/min. and regular; respirations 20/min. and regular; blood pressure 122 systolic and 76 diastolic; height 5'11"; weight 167 lbs. The patient had obvious scleral icterus. Examination of the head and neck was unremarkable. There was no lymphadenopathy. The lungs were clear to auscultation and percussion. The heart was not

enlarged and had a normal sinus rhythm with no murmurs or extra sounds. Examination of the abdomen showed no specific tenderness or palpable organs or masses. Rectal examination was negative. Examination of the extremities and neurologic examination was within normal limits.

LABORATORY DATA: Urinalysis—amber, clear; specific gravity 1.021, pH 5.0, negative for protein, glucose, ketone bodies, and hemoglobin. There was a large amount of bile. The sediment was unremarkable. Hemoglobin 14.5 gm/dl, total leukocyte count 5,900/mm³ with 44% segmented neutrophils, 2% neutrophilic bands, 12% eosinophils and 42% normal lymphocytes. The indices were within normal limits and red cells were normochromic, normocytic and platelets normal in number and morphology on the smear. Alkaline phosphatase 420 units (normal 15-65 units), heat stable alkaline phosphatase 264 units, total bilirubin 6.7 mg/dl, with 6.5 mg/dl direct and 0.2 mgs/dl indirect. Aspartate aminotransferase 273 units (normal 7-24 units), prothrombin time 12 seconds with a control of 13 seconds, total protein 9.5 gm/dl, with 4.4 gms/dl albumin, 0.4 gms/dl alpha I globulin, 1.1 gms/dl alpha II globulin, 1.6 gms/dl beta globulin, and 2.0 gms/dl gamma globulin. The alpha II globulin and gamma globulin were slightly elevated, and the electrophoretic pattern of the serum protein revealed a polyclonal type of increased gamma globulin. Leucine aminopeptidase 1,060 units (normal 75-320 units) gamma glutamyl transpeptidase 593 units (normal 8-37 units), cholesterol 296 mgs/dl, lactic dehydrogenase, calcium, inorganic phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, sodium, potassium were within normal limits. A liver scan revealed a diffusely mottled pattern throughout the liver consistent with chronic parenchymal disease. Ultrasound study revealed a hydropic gallbladder with mild dilatation of the intrahepatic biliary radicals suggesting an obstructive jaundice. The pancreas was obscured by gas in the upper abdomen. A transhepatic cholangiogram revealed non-filling of the intrahepatic biliary radicals after five puncture attempts. This was felt to indicate with 90% certainty that the patient did not have intrahepatic biliary dilatation. In 60 to 70% of normal patients with normal sized biliary tracts transhepatic cholangiography is successful. Barium enema examination showed findings consistent with chronic ulcerative colitis with a shortened bowel—lack

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of haustra in the distal two-thirds of the bowel and thickened mucosal folds on the post evacuation films. There were no active inflammatory changes. There was mild tapering and narrowing of the colon at the rectosigmoid junction. An operation was performed.

DR. SEIDEL: May we see the radiological procedures, please?

*DR. JAMES QUALE: We have barium enema films from 1957 which reveal typical changes of ulcerative colitis. There is loss of haustral pattern and shortening of the entire colon as well as diffuse superficial, small ulcerations throughout the colon. A followup barium enema in 1973 shows less ulceration but no other change.

The ultrasound examination revealed a dilated gallbladder and multiple, small lucent areas in the hilus of the liver which represent dilated bile ducts. This is consistent with extrahepatic ductal obstruction. This is as specific as one can be on this study.

At this point, I might say a few words about the radiological procedures in the evaluation of the patient with jaundice. There are three main radiographic examinations the object, of course, being to detect whether ductal obstruction is the cause of the cholestatic jaundice. These three studies are: computerized tomographic scanning of the upper abdomen (CT); ultrasonic study of upper abdomen; and cholangiography. In ultrasound and CT scanning, you can determine whether the ducts are dilated and thus have a high suspicion for extrahepatic obstruction. Usually neither of these is more specific, although occasionally a large gallstone or pancreatic mass is seen. Small lesions such as strictures of the common bile duct are not demonstrated.

The third procedure usually performed is cholangiography. Intravenous cholangiography is not done because it is rarely successful in a jaundiced patient. There are three other ways to do cholangiography. The transjugular method involves the placing of a special apparatus into the jugular vein in the neck and placing the tip of this apparatus into a hepatic vein. A needle is then placed through this and plunged into the liver in hopes of entering a biliary duct and placing contrast medium into it. Another method is endoscopic retrograde cholangiopancrea-

tography (ERCP). The other method is percutaneous cholangiography.

The percutaneous route was first instituted in Germany in 1922 but was discarded because of its complications. It was revived in the 1950's after the advent of antibiotics. It was first done by plunging a cannula with a catheter over it into the liver and withdrawing the cannula. The catheter was then slowly withdrawn hoping that it would enter a dilated bile duct. In 1974, the Japanese popularized the Chiba needle procedure. This has gained rapid popularity and is currently quite prevalent because of its low complication rate due to the small size of the needle.

Prior to the procedure, the patient is given ampicillin for systemic prophylaxis. The patient's right flank is prepped and the Chiba needle, which is a 21-23 gauge long needle, is placed into the liver with the tip located near the hilus of the liver. The stylet is taken out and the needle pulled back slowly while injecting contrast medium. A bile duct is successfully entered in nearly 100% of patients with dilated bile ducts. Normal sized bile ducts are entered in about 75% of cases. It is unusual that in this particular patient we were not successful even though the bile ducts were known to be dilated.

The complications of this procedure are below 1% and include bile leakage, bleeding, infection, pain, and fever of undetermined cause. The low complication rate with the Chiba needle contrasts with the previous complication rate of 5% with the catheter method.

Of the three studies, transhepatic cholangiography is the most definitive study. One might even consider bypassing the ultrasound and CT studies and going directly to the procedure in some cases, i.e. where it is clinically obvious that a large extrahepatic bile duct is obstructed.

**DR. KARL WEGNER: How many times do you attempt the percutaneous approach before you call the procedure unsuccessful?

DR. QUALE: The needle is passed up to five times, occasionally more.

***DR. JOHN BARKER: I don't believe you should bypass the ultrasonic studies or CT scanning particularly in patients who might have chronic liver disease. In these cases, especially with cirrhosis, it may be very difficult to penetrate the liver and enter a dilated bile duct. However, the ultrasonic studies or CT scanning may demonstrate a dilated bile duct confirming the presence of extrahepatic obstruction in addition to the chronic parenchymal disease.

Do you aspirate bile prior to injection of contrast material?

DR. QUALE: You can attempt that but you do not

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always get bile back. If you do, that is helpful.

DR. BARKER: I rarely use any antibiotic coverage when I do ERCP in spite of the fact that the instrument is not sterile. The only problem with infection occurs when you enter an obstructed area behind which infection is already present.

DR. SEIDEL: In approaching the diagnosis in this case I believe that we have to assume that the patient had extrahepatic obstruction. This is certainly indicated by the markedly elevated alkaline phosphatase and mild elevation of the transaminase. The high leucine aminopeptidase and gamma glutamyl transpeptidase and slightly elevated cholesterol certainly all corroborate the diagnosis of obstructive jaundice. The finding of dilated ducts on the ultrasonic study further suggests extrahepatic obstruction. In addition, there is no history of drug or alcohol abuse or hepatitis. The presence of a dilated gallbladder certainly is consistent with extrahepatic obstruction. A true hydrops of the gallbladder occurs when there is obstruction of the cystic duct alone and the gallbladder becomes enlarged and contains white mucus material. It is usually asymptomatic. In this case, the gallbladder is dilated along with the extrahepatic ducts as in typical extrahepatic obstruction. Therefore, I think causes of intrahepatic cholestasis such as viral hepatitis, alcoholic cirrhosis, Hodgkin's disease, sarcoidosis, and primary biliary cirrhosis are extremely unlikely in this case.

We can then concentrate on the extrahepatic causes of biliary obstruction. One must consider stones, although none are seen by ultrasonic study in the dilated gallbladder, as well as carcinoma of the head of the pancreas. These two are the most common causes of extrahepatic obstructive jaundice. Carcinoma of the bile ducts and ampulla of Vater are extremely rare. A stricture of the bile duct could produce this picture and the patient has had surgery in the region of the biliary ducts previously. With a long history of chronic ulcerative colitis one has to consider whether the patient has developed adenocarcinoma of the colon with metastasis to the lymph nodes surrounding the bile ducts with compression and obstructive jaundice. I feel that this is rather unlikely.

Dr. Seidel's Diagnosis

Extrahepatic Biliary Obstruction Due To Stones, Carcinoma Of The Pancreas

*DR. JOHN DONAHOE: Could you be more spe-

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cific as to the possible causes of obstructive jaundice in a young man who has a long standing chronic ulcerative colitis? For instance, what complications of ulcerative colitis could produce this picture of extrahepatic obstruction?

DR. SEIDEL: I would guess that one would have to consider scarring or sclerosis about the bile ducts or even carcinoma of the bile duct causing this picture.

DR. REYNOLDS: A major problem in patients with chronic ulcerative colitis is that of sclerosing cholangitis with stricture. Sclerosing cholangitis in the presence or in the absence of a history of chronic ulcerative colitis can easily be confused with sclerosing carcinoma of the bile ducts. The goals in operating on this patient were to establish a diagnosis and to relieve the biliary obstruction. Preliminary examination of the abdomen showed no evidence of tumor or metastasis. Direct exploration of the biliary duct revealed a markedly dilated gallbladder. There was marked thickening and induration of the entire common bile duct which felt like a fibrous cord or thrombosed vein fitting the description given in the literature of sclerosing cholangitis. On carefully entering the wall of this fibrous structure, we finally came to a small lumen which contained small gravel-like particles. It was not possible to pass even the smallest dilator through the lumen into the duodenum. We are very much concerned at this point whether we did indeed have a sclerosing cholangiocarcinoma. The biopsies of the lymph nodes and material from the bile duct were sent for frozen section. A diagnosis of chronic inflammation of the bile duct and a benign lymph node hyperplasia was forthcoming. A cholangiogram obtained at operation showed marked dilatation and some narrow areas scattered throughout the intrahepatic biliary tree. It was felt that because of the lack of continuity of the duct distally that a diversion procedure should be performed. We elected to do a choledochoduodenostomy. A T-tube was placed in through the cystic duct area with a long part of the tube in the proximal intrahepatic bile duct and the short part in the duodenum distally.

DR. BARLOW: Dr. Donahoe, would you say some words about the patient?

DR. DONAHOE: I saw the patient in the 1950's after he had been treated elsewhere with azulfadine for chronic ulcerative colitis for several years. He did not do well on this therapy and after about a year we decided to place him on steroids and he responded well to this therapy. It is interesting during the period of time when he was having marked problems with his disease, I had made the note that he might well come to a total colectomy. However, since the early 1960's, the patient did very well. The

patient has been well for the last 7-8 years, but in 1973 he entered with some chest pain. At that time, he was found to have no jaundice but a high alkaline phosphatase. This is why we had a liver biopsy which was reported in the protocol as a mild triaditis. The abnormal tests all reverted to normal. He did have eosinophilia at that time but his chest pain disappeared. The present admission occurred because his wife had noticed icterus in his sclerae. Because of the twenty-three year history of chronic ulcerative colitis, we were concerned about the possibility of carcinoma of the bile ducts, sclerosing cholangitis or even carcinoma of the colon with the metastasis to the porta hepatis and compression of the biliary tree.

DR. BARLOW: The material curretted from the bile duct shows chronic inflammation and fibrosis with no evidence of malignancy. (Fig. 1) A lymph node submitted with the specimen shows follicular hyperplasia and chronic lymphadenitis.



Figure 1

Material curretted from common bile duct showing chronic inflammation with no evidence of malignancy.

In addition a liver biopsy taken was histologically normal (Fig. 2). Review of the previous liver biopsy specimen showed only a very mild infiltrate of chronic inflammatory cells in the portal spaces. The finding of a normal liver biopsy is very interesting in this patient because patients with ulcerative colitis may develop severe pericholangiolitis which can lead to severe liver dysfunction and cirrhosis.

With the gross picture described by Dr. Reynolds, we have to say that this patient probably has sclerosing cholangitis. This is a rare disorder characterized by diffuse fibrous narrowing of the extrahepatic bile ducts. There may be a thickening of the gallbladder and involvement of the intrahepatic ducts as well. It has frequently been associated with ulcerative colitis but may occur as an independent entity or be

associated with other entities of unknown etiology characterized by fibrosis such as retroperitoneal fibrosis, pseudotumor of the orbit, Reidel's struma and sclerosing mediastinitis. Of course, the most common cause of stricture of the duct is previous surgery or gallstones. Pancreatitis may occasionally cause stricture of the bile ducts. Although the patient had previous surgery, it was not really in the area of the bile ducts and no stones were demonstrated other than the small gravel which Dr. Reynolds mentioned. The criteria for sclerosing cholangitis include: 1) diffuse thickening and stenosis of the extrahepatic bile ducts, 2) absence of previous biliary surgery, 3) absence of biliary tract calculi and 4) excluding carcinoma of the bile duct by sufficiently long follow-up. I think that we can safely say that the patient fits the first three criteria but sclerosing carcinoma certainly cannot be excluded in this patient as it may be very difficult to diagnose on small biopsy specimens. The differentiation of sclerosing

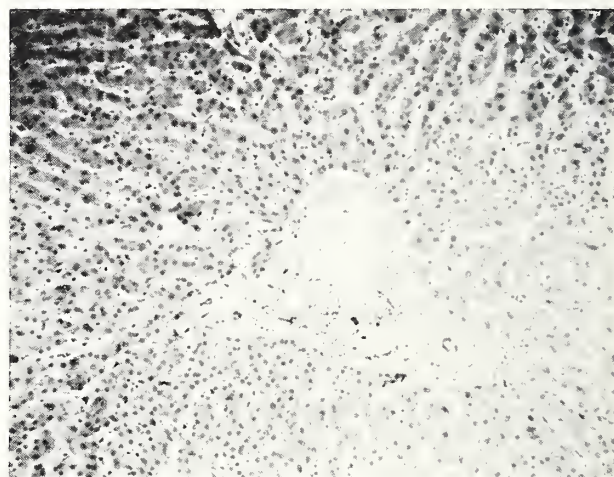


Figure 2

Histologically normal liver. The portal space shows no evidence of pericholangitis often seen in ulcerative colitis.

cholangitis and carcinoma is not merely of academic interest particularly since patients with ulcerative colitis do have an increased incidence of carcinoma of the bile ducts. This complication usually occurs many years after the colitis has been established but can even develop up to nine years after colectomy. We may have to wait several months or longer to be sure that carcinoma of the bile ducts is ruled out in this patient.

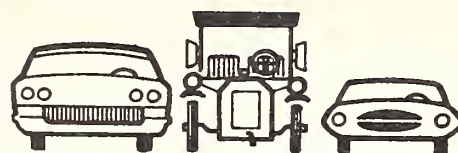
The patient certainly fits the clinical picture of sclerosing cholangitis. He had the insidious onset of obstructive jaundice with upper abdominal pain and pruritis. These patients often have hepatomegaly and may have recurrent ascending infection with hepatic abscesses or septicemia. Fortunately, the patient has not developed these latter complications. The labor-

atory can aid in making the diagnosis. This patient has had eosinophilia, hypergammaglobulinemia, an elevated erythrocyte sedimentation rate and classical liver function tests indicating obstructive liver disease. All of these have been described in sclerosing cholangitis.

FINAL ANATOMIC DIAGNOSIS SCLEROSING CHOLANGITIS

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Council Meeting Highlights

The Council of the South Dakota State Medical Association met on Saturday, January 6, 1979, at 9:30 a.m. at the Howard Johnson Motor Lodge, Sioux Falls, South Dakota. The following are major items of business transacted at this meeting.

1. Dr. Robert Ferrell was seated as Councilor from the Black Hills District Medical Society to complete the unexpired term for Dr. W. E. Jones who resigned.

2. 1979 LEGISLATIVE PROGRAM. The Council adopted the following legislative program:
Sponsored Bills:

- 1) Statute of limitations for medical corporations—2 year limit.
- 2) Statute allowing necessary medical treatment for a minor without parental consent—co-sponsor with the Hospital Association.

Endorsed Bills:

- 1) Changes in Medical Practice Act to increase fees and eliminate osteopathic examination.
- 2) \$1.1 million budget for South Dakota Human Service Center.
- 3) South Dakota School of Medicine budget request for \$820,000 plus \$100,000 for fringe benefits.
- 4) Rescind continuing education requirements for nurses.
- 5) \$250,000 request for graduate medical education—5 residency programs plus developmental monies.
- 6) License nurse practitioners in manner similar to physician assistants.

Opposed Bills:

- 1) Hospital rate review law—if one is introduced.
- 2) Expansion of Physician Assistant Law to allow four under one physician employer.
- 3) Legalization of laetrile.
- 4) Statute requiring physicians to furnish medical records to chiropractors.
- 5) Statute allowing chiropractors to perform high school physical exams.
- 6) Statute requiring Blue Shield to reimburse chiropractors.
- 7) Rescind premarital syphilis serologies.

Position to be determined by Executive Commission following the Council meeting because of inadequate information:

1) Optometry legislation.

2) Enabling legislation for Home Health Care Program.

3. PHYSICIAN OWNED INSURANCE COMPANY. Action was taken stating that the Association remain receptive to information on professional liability but not expend funds or actively pursue insurance alternatives at this time.

4. CONTINUING MEDICAL EDUCATION REQUIREMENTS FOR SDSMA MEMBERSHIP. The Council directed the executive office to draft a Bylaw change which would repeal the continuing medical education requirement of 150 hours every three years as a requirement for membership in the State Medical Association, but would support the concept of continuing medical education as a contribution to maintenance of physician competence and upgrading of quality care in the state and would encourage physicians to report accredited hours of postgraduate education to the executive office for purposes of documentation.

5. TITLE 19 (MEDICAID) CLAIM FORM. The Council approved the new Medicaid claim form as proposed.

6. DONATION TO AAMSE. The Council approved a \$100 donation to the American Association of Medical Society Executives (AAMSE) educational fund.

7. PROPOSED CHANGE IN DRIVER LICENSE REGULATIONS. Mr. Craig Kennedy, attorney at law, requested the endorsement of the Association to change driver license regulations as they pertain to restrictions for persons with seizures. The Council deferred action on this proposal pending receipt of a written draft for their consideration.

8. HOME DELIVERIES. On request of the South Dakota Board of Medical and Osteopathic Examiners the Council approved the drafting of a statement concerning home deliveries along with similar statements from the Nurses Association, the South Dakota Ob-Gyn Society, the South Dakota Pediatric Society and the South Dakota Chapter of the American Academy of Family Physicians.

9. TELEPHONE DIRECTORY LISTING BY SPECIALTY. The Council took no action on the request from Northwestern Bell Telephone Company to endorse this concept. ■

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Another use of the type and screen concerns the patient who is in danger of major hemorrhage. An example might be a patient who is relatively stable after a major gastrointestinal bleeding episode. The physician often will order a fixed number of units to be grouped, typed and crossmatched at all times. If the patient is in a precarious situation for several days, the blood assigned to him may have to be used for another patient or outdated and new units tested to replace these units. A considerable number of crossmatch changes may be accrued and the reserved blood may outdate. If the patient has a "type and screen" done, blood will be essentially available at all times under the same conditions as the surgical patient mentioned above.

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Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, i.e., dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.



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Cowboys

South Dakota is a part of an area which has been identified as being involved in the development of our western heritage. The evolution of cowboy lore and the practice of their code of ethics has been well established in this state. If you had the good fortune to have seen the television production of "Centennial" you would have been delighted to see a true "western" movie. A principle character, R. J. Poteet, personifies the epitome of the cowboy ethic much in the same way that the most revered professional and competent physician would in our society. Being uncompromising in honor and ethics was the code of the west.

I would like to suggest an author who wrote about cowboy lore with authority in his book, **Big Outfit**. Robert L. Sharp is his name and in the 1930s and 1940s his job was manager of one of the largest western ranches in North America. The location described in his book is the Southwest, first in Old Mexico and later in the northern areas of Arizona identified as the Baca Float Range. I had the pleasure of spending some time with this distinguished "old boy" down in Tucson, visiting him at his residence. He lives in an adobe built long before the turn of the century along the Tanke Verde Creek. In his book he described many of the origins of the ways of cowboys and the reasons that their life styles, customs and identity developed. He describes the "rawhide" cattle operation which infers that no help beyond supplying the habitat was provided in the raising of cattle, with the exception of the care that cowboys could render on a day to day basis watching over the vast herds. Through a period of adaptation by the livestock, success of this operation was achieved. There are some lessons to be learned from this technique which are applicable even in the days of advanced technology not only in cattle business but also numerous other industries, perhaps also in

medicine. If you are interested I may be able to obtain some copies of this interesting book of western history.*

The following poem, "The Hangman's Tree" was written to depict the physical and emotional aspects of a condemned prisoner. This particular setting was inspired by two paintings. **The Road to Nowhere** by the noted western artist, Mick Harrison,** and a smaller water color work by an artist by the name of Pechel. In the oil, **The Road to Nowhere**, a prisoner is shown, his head hung down and his hands tied behind his back, uncomfortably seated in the rear of a buckboard going his last mile across a barren sagebrush prairie accompanied by a deputy and guard in a drizzling rain.

The Hangman's Tree

Fading purple skies, slipping grey clouds
Herald the stark reality of dawn.
No moon, no hope, no song
Can help to bring life to the hangman's tree;
Or life to the man who waits
In interminable suspense, his
Spirit soon to be set free.

The cragged tree, its leaves well spent
Against the droughthy winds of the West;
Serves naught life but only death The Hang-
man's Tree.

Clanking chain imprisoned bone
Hushed against the wet brick of his last room,
Stubble beard, disheveled hair, sleepless eyes,
And repentant lies cry out
In the fading shadow of the Hangman's Tree.

Is this real? The silence screams to him
Who forgot the error of his ways.
Cannot the last mourning song of desert doves;
Their consoling call of some forsaken love
Summon a redemptive friend of the condemned?
Cannot the last chirping of August crickets
And the cacophony of locusts
Arouse some hope for life's continuum?

(continued)

* Robert L. Sharp, **Big Outfit**, University of Arizona Press.

** Authors note: It is interesting to note, for your information, that Mick Harrison, western artist has consented to illustrate this poem and others which will be a part of a publication to be bound and available in a few years. South Dakota and its western history are beautifully recalled in the works of Mick Harrison from Mobridge, SD.

The low-roofed single house in silent witness
Cries a mournful note in tune
With its own disappointed past.
Couched in the shadeless shade of
the mute terminator The Hang-
man's Tree.

Oh, what crime, what sin, has brought him
To the shade of this fateful tree?
This tree that did in life hear
The shouts and thrills of children swinging
To and fro from spring to swirling snow.

What bard will tell this futile tale?
The end in sight,
The last taut twang of rope
The last gasp of hope
Will come with dawn The Hang-
man's Tree.

Winston Bryant Odland ©

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Cardiology Today, U. of Iowa, Iowa City, IA, May 14-17. AMA Category I and AAFP credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Marriage and Divorce Counseling, V. A. Hospital, Fort Meade, SD, May 17. 1 hr. AMA Category I credit. Contact: Office of Cont. Health Ed., USD, Sioux Falls, SD 57105, (605) 339-7573.

Pain: Diagnosis and Treatment of the Difficult Patient, McKennan Hospital, Sioux Falls, SD, May 19. 8 hrs. AMA Category I credits. Contact: Bill Arbes, Ph.D., Dept. of Psychiatry, USD School of Medicine, Sioux Falls, SD 57105, (605) 339-6785.

Second National Conference on Genetics and the Law, Copley Plaza Hotel, Boston, MA, May 21-23. Fee: \$50. Contact: Aubrey Milunsky, M.D., 200 Trapelo Rd., Waltham, MA 02154.

Family Practice Review: Update 1979, Radisson-St. Paul. St. Paul MN., May 21-25. Fee: \$275. Contact: Office of CME, Box 293 Mayo Memorial Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

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Current Concepts in Radiation Therapy, Mayo Memorial Aud., U. of Minn., Minneapolis, MN, May 23-25. Fee: \$200. Contact: Office of CME, Box 293, Mayo Memorial Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Laboratory Medicine Update 1979, IDS Center Conf. Theatre, Minneapolis, MN, May 23-25. Fee: \$200. Contact: Office of CME, Box 293 Mayo Memorial Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Gastric Bypass, U. of Iowa, Iowa City, IA, May 24-25. AMA Category I and AAFP credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

June

South Dakota State Medical Association Annual Meeting, Howard Johnson Motor Lodge, Rapid City, SD, June 8-10. Contact: SDSMA, 608 West Ave., North, Sioux Falls, SD 57104.

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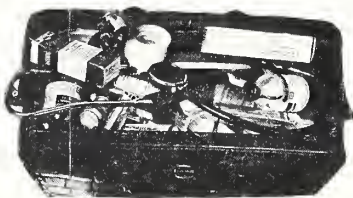
Fourteenth Annual Meeting of the Rocky Mountain Neurosurgical Society, Jackson Lake Lodge, Jackson Hole, WY, June 13-17. Contact: Ralph J. Kaplan, M.D., Sec., U. of Oklahoma Health Sciences, Center, P. O. Box 25606, Oklahoma City, OK 73125.

10th International Congress for Suicide Prevention and Crisis Intervention, Ottawa, Ontario, Canada, June 17-20. Fee: \$125. Contact: Secretariat, IASP Congress, Suite 700, 71 Bank St., Ontario, Canada K1P 5N2.

Scientific Seminar on Advances in Pediatrics, Sylvan Lake, Custer, SD, June 20-22. AMA Category I and AAFP credits. Contact: Thomas Aceto, Jr., M.D., Dept. of Pediatrics, U. of S.D. School of Med., McKennan Hosp., Sioux Falls, SD 57101.

Northern Michigan Summer Program, Shanty Creek Lodge, Bellaire, MI, June 25-29. Contact: Office of CME, U. of Michigan Med. School, Towsley Center for CME, Ann Arbor, MI 48109.

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Widows And Widowhood

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Table of Contents: page 3

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Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
22	10	1 teasp. (5 ml)	½ tablet
44	20	2 teasp. (10 ml)	1 tablet
66	30	3 teasp. (15 ml)	1½ tablets
88	40	4 teasp. (20 ml)	2 tablets or 1 DS tablet

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SCIENTIFIC ARTICLES

- 9 Widows And Widowhood
David W. Bean, M.D.
Cecilia M. Roberts, Ph.D.

- 24 X-Ray Case Of The Month
Donald J. Peik, M.D.
Martin F. Petereit, M.D.

- 33 Clinicopathological Conference
Thirty-Five Year Old Man With Swelling Of The
Right Testis
Dennis Foster, M.D.
John Hoskins, M.D.
John F. Barlow, M.D.

FEATURES

- 16 Laboratory Aids
South Dakota Pathology Page
- 18 South Dakota AFP Chapter News
- 21 President's Page
- 23 This Is Your Medical Association
- 43 Future Meetings

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Widows And Widowhood

David W. Bean, M.D.*

Cecilia M. Roberts, Ph.D.*

ABSTRACT

Physicians, even those skilled in dealing with dying patients, often find difficulty providing useful support to those of their patients experiencing the phenomenon called widowhood. These patients experience a host of physiological and psychological sequelae and the physician's ability to assist them depends on his or her understanding of the previous marriage relationship, the physiological risks in

Physicians, even those skilled in dealing with dying patients, often find difficulty providing useful support to those of their patients experiencing the phenomenon called widowhood. These patients experience a host of physiological and psychological sequelae, and the physician's ability to assist them depends on his or her understanding of the previous marriage relationship, the physiological risks in widowhood, and methods of coping with loss and bereavement.

MARRIAGE

Marriage is a joining of two persons of the opposite sex in a limited, mutual, nonaggressive pact in an effort to better support themselves in their attempts at coping with life. A prominent purpose of this relationship is to provide individual psychological need fulfillment through mutual support. A good marriage does exactly that—each partner's needs are satisfied through his/her relationship with the other partner.

As these relationships progress in depth as well

widowhood, and methods of coping with loss and bereavement.

Human beings have characteristic reactions to psychological losses; these reactions include denial, anger, bargaining, depression, and finally acceptance. In addition, bereaved persons have measurably increased risk of physiological illness.

The authors offer suggestions to physicians seeking to help their bereaved patients.

as in time, the two persons become less differentiated as individuals and more "one" in a real psychological sense. Individual independence is gradually replaced by mutual dependency and two persons become one couple. This coupling, while providing mutual strength, has the hazard or rupture through death of one of the members with a resulting unsupported dependency state left unfulfilled in the remaining person. This is a real and significant loss.

PSYCHOLOGICAL REACTIONS TO LOSS

Human beings have characteristic reactions to psychological losses be they small or large, and the loss of a mate is exceptional only in degree and not in kind.

The first reaction is **shock and disbelief**. It is much like the experience of being hit in the head with a hammer. The patient feels stunned, and can't believe it is happening to her. This stage, which Kübler-Ross¹ calls denial, provides a cushion of time during which the new widow may grow gradually to experience her loss. Denial may be longer if death was unexpected, was unwitnessed, and/or the body was destroyed.

Second, there is a reaction of anger, and a questioning of why is this happening to me? This anger

* Professor and Chairman, Dept. of Psychiatry, USD School of Medicine.

**Associate Professor, Dept. of Psychiatry, USD School of Medicine.

may be directed at fate, God, the husband who has abandoned her, and, last but not least, the doctor. (Don't be upset, they still love you even though angry! You must be important for your patient to be so angry with you.)

The third of Kübler-Ross' stages, that of bargaining, takes on a different character in bereavement than it has with the dying patient. On closer analysis, however, both are founded on hope. The new widow says, in essence, "YES, HE IS DEAD. But he can still hear me, see me, and I will see him again." We commonly hear this sort of bargaining referred to as "unrealistic hope". The adjective is redundant; **all** hope is unrealistic. We hope for what we haven't got, not for what we have, and unless the hope is so unrealistic that it damages the widow or others (refusing to bury the dead body, carrying on conversations with the departed in the presence of grieving children) it should be tolerated and welcomed. It's useful—that's why she's using it.

Then follows a significant grieving reaction, a reactive depression with all the usual signs—sleep disturbance, loss of appetite, tears, sighing, etc.

Finally, if all goes well, with time and help the widow reaches acceptance and reconstitutes herself into a functioning individual carrying on with life's everlasting problems.

Obviously, each widow will react to her loss depending on a number of variables including her own personal strength, etc., but the general reaction follows this pattern.

The following may more graphically portray the widow's experience, and might be entitled, "How You Feel When You Found Out Part of You Died A Week Ago." It goes like this:

1. Exhaustion—"Oh, how tired I am. No matter how much I rest or sleep I feel exhausted all of the time."
2. Lethargy—"My limbs are so heavy I feel as though I'm walking through molasses—in January, no less."
3. Loneliness—"I feel so alone, as though no other person in this whole world cares for me. I feel like an abandoned child standing in the middle of a dusty road."
4. Anger—"I feel an undercurrent of bubbling and even raging anger which I cannot express. If I could, I surely would get that person who did this to me!"
5. Confusion—"But I just don't know who did this to me—or why, I just can't make any sense of it."
6. Hopeless—"With all these feelings I have, it just doesn't seem like there is any reason to go on—I would if I could, but I just see no

way out."

7. Defenseless—"If all that isn't enough, I find that all that I depended upon is now gone! I'm standing here naked, shivering, crying and, oh, what am I to do? Won't someone, somewhere, help me!"

PHYSIOLOGICAL REACTION

Mortality and morbidity figures tell us that new widows (and new widowers) are at increased risk in many areas. An increased death rate for surviving spouses peaks in the first six months following the bereavement (various sources suggest an increase of 17% to 40%) and returns to normal at about one year. In one study, three-quarters of these excess deaths were attributed to heart disease, largely coronary thrombosis and arteriosclerotic heart disease.²

High rates of recent widowhood are also associated with leukemia, ulcerative colitis, asthma, and possibly with cervical cancer. More widows than non-widows consult physicians for arthritis and rheumatic conditions, and among those under 65 more widows than non-widows seek help for anxiety, depression, and insomnia. Other symptoms reported more by widows include headaches, dizziness, fainting spells, blurred vision, skin rashes, excessive sweating, indigestion, difficulty swallowing, vomiting, heavy menstrual periods, palpitation, chest pain, shortness of breath, frequent infections, and general aching.

Widows have been found to consume more alcohol, tobacco and tranquilizers after their bereavement than before, and are, as a group, hospitalized more frequently than are non-widows. Seeking help for their problems, they turn to chemicals, clergymen, psychiatrists and social workers; the major study of American widows shows that they do **not** often seek help from their physician.³

A simple explanation for their latter phenomenon may be financial; widowhood usually imposes reduced income, and health insurance typically covers hospitalization but not consultation with the physician. In mid-America, then, the new widow may have few options. Psychiatrists and social workers don't grow on trees on the prairie, and ministers like physicians may want to help but not know how.

WHAT CAN PHYSICIANS DO TO HELP

The physician who wants to help his widowed patient may find the following "helpful hints" of value. None is difficult, none requires special talent or special training. All take time, the time of a busy physician for whom time is money.

1. Offer your support to the widow. She may not know how to use it initially, but she will appreciate the offer and the offer itself will be

of great assistance.

2. Describe the nature of "loss" phenomena so that they will know what to expect. Knowledge is a fantastic antianxiety agent.
3. Allow them to ventilate their feelings fully to you without moral approbation as to the nature of their feelings. This is especially true of the patient's anger. They will be "mad like hell" at the mate who left them alone. This anger is not a lack of love but really a testimony to the need for the now lost mate. It is often helpful to inform the angry widow that the opposite of love is not anger but apathy, and she should feel no real guilt at being angry at her loss.
4. Inform patients that their feelings are not abnormal and that they are not immoral persons for having such feelings. They have enough to cope with without having to deal with unnecessary fears and guilts.
5. Call in external support systems such as family, friends, ministers, etc., to help support the patient's now unfulfilled dependency needs. It is surprising to find how many external people would like to help but "don't wish to be a bother". Encourage their involvement.
6. As the process progresses, encourage the patient to develop new interests and activities and, more particularly, encourage participation in the so-called self-help groups. Widow's clubs are of significant use and support to these persons, for they help in expressing feelings, provide mutual aid and support and, generally, have a plethora of information on "how to get along" when alone.
7. Finally, caution your patient against "blind alleys". At least a few widows in their attempts to cope with their loss, and in response to their loneliness, seem to be especially susceptible to the following:
 - a. Get rich schemes—It does seem that the jackals of our society enjoy preying on a widow's financial worries to ease her out of whatever she has left, financially, by various confidence games.
 - b. The drink a lot club—Alcohol is a wonderful antidepressant and antianxiety agent—except that it rots the brain and the rest of the body. So for goodness sake, warn your patients of this trap and, if they seem to be traveling this trail, refer them to good psychiatric help.
 - c. The go to bed a lot club—When a woman is lonely and abandoned she may occasionally try to "cure" her problem by sexually

bouncing from bed to bed even though she may acknowledge that it is really not very helpful. This may represent a feeling that "bad breath" is better than no breath at all—but it is terribly self-destructive and these women do not need any more destruction in their lives.

- d. The rebound marriage phenomenon—Some women, in an effort to cure their pain, seem to grab any man that comes along to mate with in some faint hope that this will reincarnate the lost mate and fill their dependency void. The old story of the rebound marriage by the jilted lover is an old folk tale with a real moral to the story, and widows are ill advised to remarry too suddenly.

This is **not** to say that widows should not remarry. Having accomplished a previously satisfactory marital relationship makes them good candidates for another satisfactory relationship, provided the new marriage isn't grounded only in "rebound phenomenon."

In summary, to be of real assistance to the widow, be kind, gentle and caring. Remember "Old Doc" whom everyone loved? He didn't cure a lot of patients, but he did care. Let us cure when we can, but never stop caring for our patients.

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...in the functional bowel/irritable bowel syndrome*

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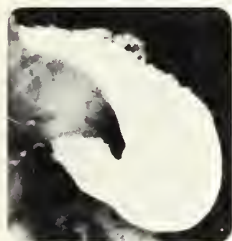
10 mg. capsules, 20 mg. tablets,
10 mg./5 ml. syrup, 10 mg./ml. injection

helps control abnormal motor activity
with minimal anticholinergic side effects[†]

Demonstrated smooth muscle relaxant activity.

In this double-blind study, twenty patients having G.I. series and exhibiting spasm were randomly selected to receive either 2 cc. of Bentyl or sodium chloride intramuscularly. Ten minutes after the injection another radiograph was taken . . .

. . . Bentyl produced definite relaxation in 8 of 10 patients. The sodium chloride produced relaxation in only 3 of 10. No side effects occurred in either group of patients.



Pylorospasm has almost totally blocked passage of barium meal.



Barium meal beginning to pass 10 minutes after intramuscular injection of 20 mg. Bentyl.

"The correlation of spasm relief and drug given was excellent."

*This drug has been classified "probably" effective in treating functional bowel/irritable bowel syndrome.

†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:

King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

Merrell

Bentyl[®]

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AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

INDICATIONS

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the following indications as "probably" effective:

For the treatment of functional bowel/irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

THESE FUNCTIONAL DISORDERS ARE OFTEN RELIEVED BY VARYING COMBINATIONS OF SEDATIVE, REASSURANCE, PHYSICIAN INTEREST, AMELIORATION OF ENVIRONMENTAL FACTORS.

For use in the treatment of infant colic (syrup).

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloroduodenal stenosis), paralytic ileus, intestinal atony of the elderly or debilitated patient; unstable cardiovascular status in acute hemorrhage; severe ulcerative colitis; toxic megacolon complicating ulcerative colitis; myasthenia gravis. **WARNINGS:** In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. **PRECAUTIONS:** Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy. Use with caution in patients with: Autonomic neuropathy. Hepatic or renal disease. Ulcerative colitis. Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon. Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension. Hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.

Do not rely on the use of the drug in the presence of complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. **ADVERSE REACTIONS:** Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia; urinary hesitancy and retention; blurred vision and tachycardia; palpitations; mydriasis; cycloplegia; increased ocular tension; loss of taste; headache; nervousness; drowsiness; weakness; dizziness; insomnia; nausea; vomiting; impotence; suppression of lactation; constipation; bloated feeling; severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating. With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. **DOSEAGE AND ADMINISTRATION:** Dosage must be adjusted to individual patient's needs.

Usual Dosage: Bentyl 10 mg. capsule and syrup: *Adults:* 1 or 2 capsules or teaspoonfuls syrup three or four times daily. *Children:* 1 capsule or teaspoonful syrup three or four times daily. *Infants:* ½ teaspoonful syrup three or four times daily. (May be diluted with equal volume of water.) Bentyl 20 mg.: *Adults:* 1 tablet three or four times daily. Bentyl Injection: *Adults:* 2 ml. (20 mg.) every four to six hours intramuscularly only. **NOT FOR INTRAVENOUS USE. MANAGEMENT OF OVERDOSE:** The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal. Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested. If indicated, parenteral cholinergic agents such as Urecholine[®] (bethanecol chloride USP) should be used.

Product Information as of October, 1978.

Injectable dosage forms manufactured by CONNAUGHT LABORATORIES, INC., Swiftwater, Pennsylvania 18370 or TAYLOR PHARMACAL COMPANY, Decatur, Illinois 62525 for MERRELL-NATIONAL LABORATORIES, Division of Richardson-Merrell Inc., Cincinnati, Ohio 45215, U.S.A.

S D

Laboratory Aids

Sponsored by the South Dakota Society of Pathologists

Proper Use of the Radioiodine Uptake (RAIU)

With much more accurate, precise, and more easily performed in vitro determinations of thyroid function, the RAIU has become a less useful test for thyroid function. However, the RAIU is extremely valuable in determining the cause of and in selecting appropriate therapy in cases of hyperthyroidism.

The most common cause of hyperthyroidism is diffuse hyperplasia (Grave's Disease). The RAIU is usually elevated and the scan shows diffuse hyperplasia. Single or multiple toxic adenomas may cause hyperthyroidism. The scan is typical and the RAIU is normal or elevated. Other rare types of hyperthyroidism such as those caused by pituitary tumors producing thyroid stimulating hormone are also associated with elevated RAIU values.

However, there are several conditions with thyrotoxicosis accompanied by low RAIU values. One of the most important of these is a form of thyroiditis (probably viral in origin) which produces self limited hyperthyroidism often followed by transient hypothyroidism and a subsequent return to the normal euthyroid condition in a matter of few months. The gland is often painful, enlarged and tender. However, all of these signs or symptoms may be absent, but the patient still may manifest hyperthyroidism as a presenting condition. An important clue to the diagnosis is the very low value of the RAIU. Antibody titers to thyroglobulin and microsomal fraction are also low. The disease is self limited and can be treated with propranolol. Ablative surgical or radiation therapy are unnecessary and antithyroid drugs to stop production of thyroid hormone are not effective since the hormone release is on the basis of the inflammatory process. Propranolol therapy is sufficient. Thyroiditis has been estimated to cause 4-16% of the cases of thyrotoxicosis.

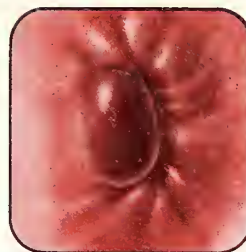
Other causes of hyperthyroidism with low RAIU values include thyrotoxicosis factitia (surreptitious use of thyroid hormone), hyperfunctioning struma ovarii, hyperfunctioning metastatic thyroid carcinoma and excessive ingestion of iodide (Jod-Basedow's Disease). History, physical examination and appropriate scan or x-ray examination can often lead to the correct diagnosis and proper therapy in these conditions.

John F. Barlow, M.D.
Pathologist

Merrell

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For hemorrhoids and other anorectal conditions



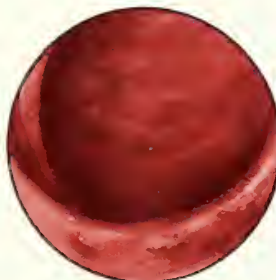
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Pruritus ani



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CAUTION: Federal law prohibits dispensing without prescription.

Description: Each Anusol-HC Suppository contains hydrocortisone acetate, 10.0 mg; bismuth subgallate, 2.25%; bismuth resorcin compound, 1.75%; benzyl benzoate, 1.2%; Peruvian balsam, 1.8%; zinc oxide, 11.0%; also contains the following inactive ingredients: bismuth subiodide, calcium phosphate, and certified coloring in a hydrogenated vegetable oil base.

Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, bismuth subiodide, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Indications: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas and relief of local pain and discomfort following anorectal surgery.

Anusol-HC Cream is also indicated for pruritus ani. Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol[®] Suppositories or Ointment.

Contraindications: Anusol-HC[®] Suppositories and Anusol-HC[®] Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparation.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts, or for prolonged periods of time.

Precautions: Symptomatic relief should not delay definitive diagnoses or treatment. If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Core should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Anusol-HC is not for ophthalmic use.

Dosage and Administration: Anusol-HC Suppositories—Adults: Remove foil wrapper and insert suppository into the anus. One suppository in the morning

and one at bedtime, for 3 to 6 days or until inflammation subsides. Then maintain patient comfort with regular Anusol Suppositories.

Anusol-HC Cream—Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the external surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain patient comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories—boxes of 12 (N 0047-0089-12) and 24 (N 0047-0089-24), in silver foil strips with Anusol-HC W/C printed in black.

Anusol-HC Cream—one-ounce tube (N 0047-0090-01); with plastic applicator, detachable label.

Store between 15°-30° C (59°-86° F).

Full information is available on request.



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Tenth Anniversary of Family Practice as Medical Specialty

The medical specialty of family practice, which evolved from general practice and whose professional ancestor was the time-honored family doctor, was 10 years old on February 8, 1979.

During that decade, more than 19,000 family doctors have been certified in this specialty by virtue of taking a special two-day examination to prove their competence. More than 6,000 young doctors are undergoing family practice training in three-year residency programs. Over 1,800 graduates of these approved training programs now are in practice throughout the United States, a large percentage in towns under 30,000 population.

The family physician, who provides comprehensive, continuing care and serves as health advocate to the entire family, is on the way back after years in the shadow of the superspecialist in a fragmented system. Prevention of disease and the partnership of patient and physician are hallmarks of his/her type of practice. The family practice approach embraces the time-honored "house call," when necessary and useful, and focuses on the patient as a person instead of a system of organs or a disease entity.

The specialty of family practice, a specialty in breadth rather than depth, was born with the creation of the American Board of Family Practice (ABFP) on February 8, 1969, at the Conference on Medical Education at the Palmer House hotel in Chicago. The conference, an annual event sponsored by the American Medical Association (AMA), brought together medical educators from all over the country.

There now are 19,144 diplomates, all of whom were required to pass the exam. The ABFP has proved to be a pacesetter as well as being highly innovative in its role of a competence measurer and credentials guardian. It was the first specialty board to have mandatory recertification, requiring recertification by examination every six years. It also was the only specialty board to require examination; other specialties had permitted "grandfathering," or certification by fiat, for those practicing the specialty when the board was established. The ABFP is the only certifying board that incorporates other types of specialists in the board of directors—regular members of its 15-member board represent internal medicine, pediatrics, surgery, obstetrics/gynecology, and psychiatry/neurology, as well as family practice.

— — —

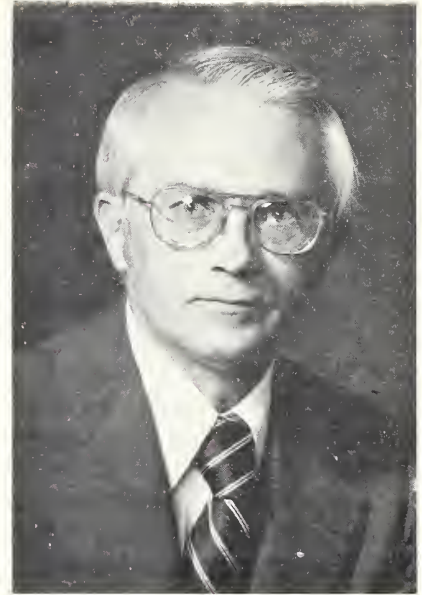
From ANGEL OF BETHESDA, by Cotton Mather, 18th century American

"I will in the first place readily acknowledge that one of the worst Maladies, which a Man in any Trade, or Way of Living, can ever fall into is for a Man to be sick of his Trade. If a man has a Disaffection to the Business that he has been brought up to and must live upon, 'tis what will expose him to many and grievous Temptations and hold him in a sort of perpetual Imprisonment. Man, beg of God a heart reconciled unto thy Business, and if He has bestow'd such a Heart upon thee, as to take Delight in thy daily Labour, be very thankful for such a Mercy!"

— — —

Black Hills Summer Seminar

Mark your calendar for August 9-11. First mailing in May.



The economic control of medical care by government has received much promotion and publicity in recent times, and most of us have felt this would, if it should come to pass, be to the detriment of the public. It could do nothing but breed total mediocrity into the health care system. Now, beginning in earnest, these same elements are dropping the other shoe.

Proposals in many forms from different levels of government are being proffered to control the *standards* of the medical profession. The Joint Commission on Accreditation of Hospitals is forcing more and more paper work on the profession that only in the most remote sense, has any effect on what the patient gets for his money in the form of good care. This seems almost a knee-jerk response to government threat of taking over their accreditation function if they don't adhere to the bureaucracies idea of "tightening things up." PSROs were formed for "quality assurance" to be certain that physicians were performing their jobs according to "acceptable standards" and laying out a basic cookbook form to be followed for every diagnosis. The Federal Trade Commission is attacking the Liaison Committee for Medical Education (the accrediting body for medical schools) with the implication they cannot be trusted and a government agency should do it for the benefit of the people. The Federal Trade Commission is threatening the

American Society of Plastic and Reconstructive Surgeons for their requirement of Board certification for membership, stating the requirement might constitute unfair restraint of trade. This would, of course, imply opening the field to lesser trained people, while at the same time, a congressional subcommittee has said congress should consider legislation to develop "minimum standards for competency" of physicians performing surgery.

The ploy, obviously, is to first destroy the present structure and then have government replace it with one of their own design. The double thrust of government to control medical practice and medical care specifically is alluded to in a 1961 pamphlet entitled "The Case for Socialized Medicine." In part, it stated "We can do everything possible to encourage Federal intervention in the financing of *medical costs* on a bit by bit basis..... We must give high priority to attempts to amend legislative proposals in ways that will involve the government in *medical standards*....."

If government continues to be successful in these endeavors, medicine will no longer be a profession. It will be a trade and I cannot imagine how this will *benefit* the people of our country.

Fraternally yours,
Russell H. Harris, M.D., President
South Dakota State Medical Association

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SD

This Is Your Medical Association

Governor Janklow appointed **G. Robert Bartron, M.D.**, Watertown, to the Board of Regents to complete the unexpired term of David Morrill which ends January 1, 1981.

* * * *

James Larson, M.D., Watertown, has been elected to the Board of Directors of Memorial Medical Center, Watertown, SD. **S. W. Allen, Jr., M.D.**, was re-elected president of the Board.

* * *

Officers elected to the Dakota Midland Hospital Board of Governors, Aberdeen, include **Paul Leon, M.D.**, president; **William Bormes, M.D.**, vice president; and **Juan Chavier, M.D.**, secretary-treasurer.

* * * *

Robert J. Pelegrin, M.D. has opened his office for the practice of urology in Huron. Dr. Pelegrin is a graduate of the University of Iowa. He served his internship at Springfield City Hospital, Springfield, Ohio, and then practiced in Davenport, Iowa for four years. He served a surgical residency at the V.A. Hospital, Leavenworth, Kansas and a urology residency at the University of Missouri, Kansas City. He then practiced in Independence, Missouri for five years prior to coming to Huron. His office is located at 66 Third St., S.E., Huron, SD.

Morris C. Benson, M.D. has joined **C. A. Johnson, M.D.**, in the general practice of medicine at the Five Counties Clinic, Lemmon. Dr. Benson graduated from the University of Calgary in 1976, he served a surgical internship at Strong Memorial Hospital, Rochester, New York, from 1976-1977, and a thoracic and cardiovascular surgery residency at University Hospital, Edmonton, Alberta, Canada, from 1977-78. Prior to moving to Lemmon Dr. Benson was in private practice in Saskatchewan, Canada.

* * * *

Thomas Henry, M.D., Rapid City, presented a program on autopsies and causes of death at the monthly meeting of the Rapid City Jaycettes.

Karl Kosse, M.D., Aberdeen, has been elected president of the St. Luke's Hospital staff. **James Hovland, M.D.** was elected vice president and **Randy Heisinger, M.D.** was elected secretary.

* * * *

The Watertown District Medical Society elected **W. N. Guddal, M.D.**, president for 1979. Other officers are **B. J. DeSai, M.D.**, vice president and **Gerald Tracy, M.D.**, secretary.

* * * *

The South Dakota Psychiatric Association held its organization meeting and elected the following officers for 1979: **David Bean, M.D.**, Yankton, president; **William Fuller, M.D.**, Sioux Falls, vice president; **David Gehlhoff, M.D.**, Sioux Falls, representative; **Carl Rutt, M.D.**, Sioux Falls, deputy representative and **Daniel Kennelly, M.D.**, Sioux Falls, secretary-treasurer.

* * * *

**YOUR
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FUND**

Merwin Lanam, M.D., former South Dakota physician, died at age 80 in Sun City, Arizona. He graduated from the University of South Dakota School of Medicine and Rush Medical College in 1928. He practiced general medicine and surgery in Sioux Falls for 23 years and in Rapid City for 10 years. Survivors include his wife, Francis, two daughters and two sons, and 15 grandchildren.

SD

X-Ray Case Of The Month



Martin F. Petereit, M.D.*

Donald J. Peik, M.D.*

Author's Note

About ten years ago, one of us (M.F.P.) authored a number of "Radiologic Case Presentations" which continued for about five years. The emphasis was more upon unusual, but not necessarily rare situations. Over the years, many publications have had "case of the month" type of presentations, offering a type of stimulus, as well as, useful information. Similar objectives inspired a similar type of series for this journal. However, for a number of reasons, including personal inertia, the series died!

Dr. Peik and I have decided to start a new series, but with an entirely different format. The presentations will be shorter, the photographs larger, and most of the cases will be reasonably common. The emphasis will be on the "basics" of x-ray diagnosis. Dr. Peik and I will alternate preparation of the cases, but will co-author them. At times, other members of our group may elect to contribute a special interest topic. Dr. Peik, whose hobby is

photography, will prepare the photographs for these presentations.

The field of radiology continues to be involved with progressive changes. An incredible number of signs have been described to enhance the value of this diagnostic modality. Some of these signs may be very subtle and yet have very serious implications. At times, the authors may stress certain views or examinations, not only as an instructive aid, but also to promote an increase in understanding between clinicians and radiologists in general.

As before, the suggested approach is: (1) view the entire film (2) attempt to discover the key findings (3) list a differential diagnosis, with the most likely possibility at the top of the list, and then read the description. The authors invite any comments and/or suggestions from the readers. Initially these presentations will appear four times per year. Thank you.

CASE

Figure 1 is a portable anteroposterior 40 inch chest film of a male in his 50's involved in a severe motor vehicle accident (MVA), having collided with a telephone pole. He was brought to the emergency room at Sioux Valley Hospital. The chest film was soon taken. How many positive findings are there? What is the most obvious finding? Which is the most serious? Based on the most serious finding, what would be your next step?

Positive findings include interstitial air along the left chest wall, fractured left ribs, hazy alveolar infiltrate left upper lung, and **more important,**

widening of the upper mediastinum, deviation of the tracheal air column to the right, and a poorly defined aortic shadow. With the history of severe deceleration injury, and the chest film findings, the possibility of traumatic aortic rupture (TAR) was strongly considered. The patient's clinical condition did not permit time for angiographic evaluation. He was taken to the operating room, where he expired. Post-mortem examination revealed aortic transection at the level of the isthmus. The time lapse from injury to death was just a few hours.

DISCUSSION

One of the first questions to be answered from the chest film is: is there a mediastinal hematoma? Remember that often these chest films are taken with portable technique, which usually means AP

*Radiologists, Medical X-ray Center, Sioux Falls, SD.

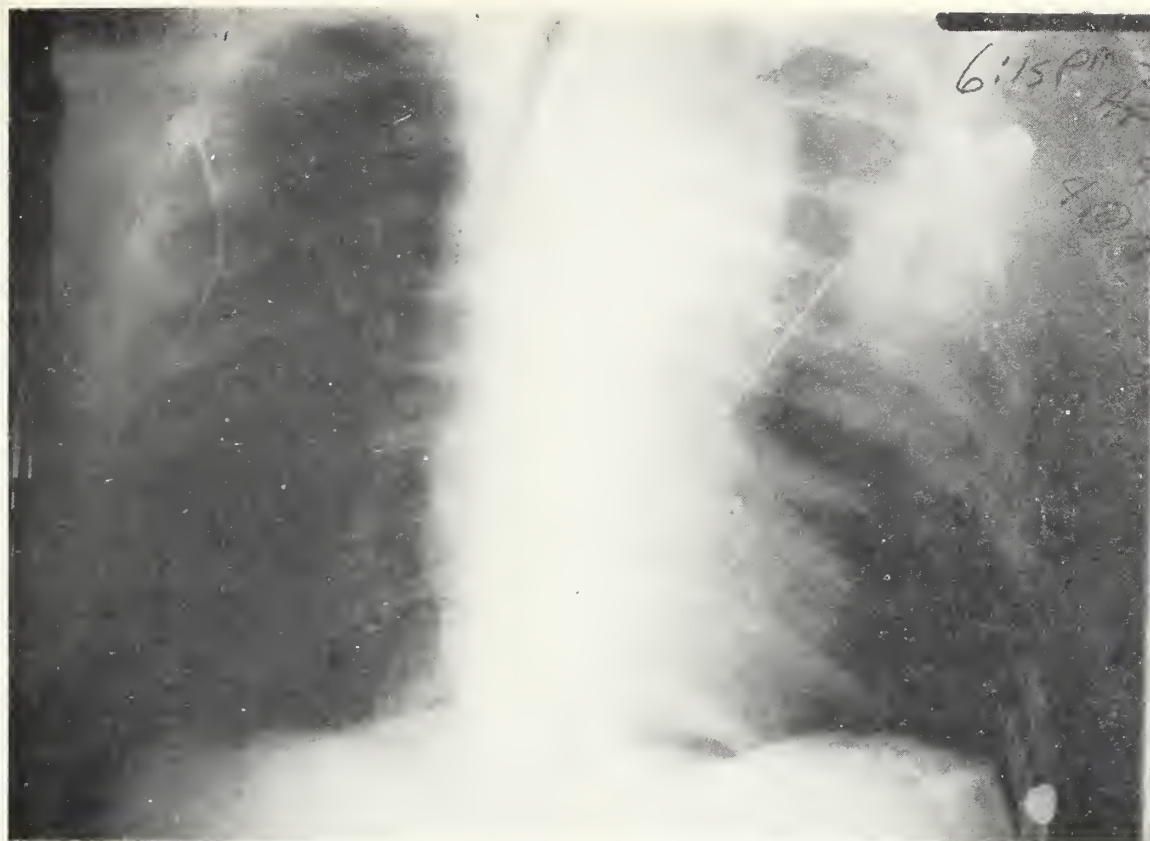


Figure 1

40 inch supine. Both the shorter distance and the AP projection contribute to more **magnification** and this can make a normal mediastinum appear abnormally wide. Mediastinal hematoma is due to localized hemorrhage and may be secondary to chest cage compression or deceleration injury. It is fairly common with pulmonary contusion, and is less often due to TAR. Probably many cases go unrecognized, since the amount of bleeding may be insufficient to produce symptoms. Signs and symptoms are often not striking. Mediastinal hematoma should be suspected when there is retrosternal pain radiating to the back. The mediastinal widening may be symmetric or asymmetric. If there is true widening of the upper mediastinum on a post-traumatic chest film, angiography is mandatory to evaluate the possibility of TAR!

If there is a mediastinal hematoma, the next question to be answered is: is there great vessel injury? The main consideration is the aorta. Of all deaths caused by auto accidents, 15-20% are secondary to TAR. The **isthmus** (slightly distal to the origin of the left subclavian artery and site of ligamentum arteriosum) ruptures three times as often as the ascending or descending segments, because the aortic arch is immobilized by the brachiocephalic vessels, whereas the descending aorta

is relatively mobile. Thus, the junction of these two segments, the isthmus, is more susceptible to rupture in injuries involving sudden deceleration. Eighty per cent of patients with tears at the isthmus are dead on arrival at the hospital. Out of the remaining 20%, 9 out of 10 survive longer than 6 hours. About 5% will live to develop a chronic aneurysm (almost all of these are at the isthmus). This is a pseudoaneurysm, since the wall of it is composed of mediastinal connective tissue. TAR is uncommon in children, probably due to a cushioning effect of the thoracic cage with its greater elasticity.

According to one author, about one third of patients with TAR have little or no evidence of chest trauma at the time of the initial physical examination. Also, many will have little significant findings on the initial chest film. Therefore, if TAR is suspected, frequent chest films should be taken, especially the first 48 hours. However, some feel that if TAR is even remotely suspected, aortography should be performed. Complete transection occurs in up to 40% of all aortic ruptures. Complete transection is not incompatible with survival; in some cases, blood is contained by the adventitia and surrounding tissues.

Plain film findings include: tracheal deviation to the right (provided patient has the usual left sided

aortic knob!), increased width of upper mediastinum, loss of aortic knob shadow, left sided pleural fluid (blood), rib fractures, depression of left main stem bronchus, pneumothorax, pneumomediastinum, and pulmonary contusion. However, the two most valuable signs are increased width of mediastinum and deviation of trachea.

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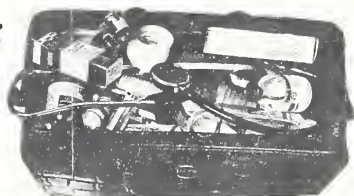
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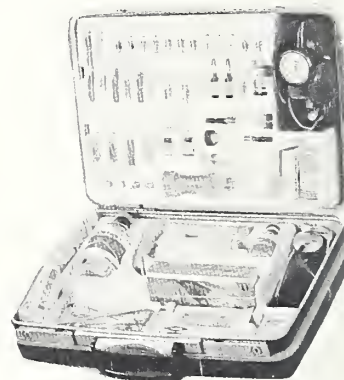
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Thirty-Five Year Old Man With Swelling Of The Right Testis

Dennis Foster, M.D.*

John Hoskins, M.D.**

Discussers

John F. Barlow, M.D.***

Editor

Case No. 758285

This 35-year-old man was referred to Sioux Valley Hospital because of painless swelling of the right testis.

The patient had been perfectly well until three days prior to admission when he noticed painless firm swelling of the right testicle unaccompanied by redness and with no history of trauma. The testes had been descended since birth. The patient gave no history of past surgery except for removal of tonsils and adenoids as a child. There was no history of hypertension and the review of systems was negative.

PHYSICAL EXAMINATION: The patient was alert and in no distress. Temperature 98.8°F, pulse 86 per minute and regular, respirations 20 per minute and regular, blood pressure 144 systolic and 82 diastolic. Height 6'1", weight 214 pounds. Examination of the head and neck was unremarkable. The chest was clear to auscultation and percussion. Heart was within normal limits of size with no murmurs and had a normal sinus rhythm. The abdomen showed the liver to be 3 cm. below the costal margin but appeared to be normal in width to percussion. There were no nodules felt. There were no other organs, masses, or tenderness in the abdomen. Examination of the genitalia showed a normal appearing penis and both testes were descended and generally of comparable size. The left testis was firm with a normal configuration. The epididymis felt normal. The right testis was extremely firm but maintained a generally normal outline and was slightly broader in the lower pole. It was difficult to differentiate the testis from the epididymis.

LABORATORY DATA: Urinalysis clear, straw colored, specific gravity 1.028, pH 5.0, negative for protein, glucose, ketone bodies, bile, and hemoglobin; sediment 0 to 2 white

cells per high power field, no red cells and no casts. There were a few bacteria and a few mucus threads. Hemoglobin 16.4 gm/dl, red count 5.5 million/mm³, hematocrit 49 vol. %, mean corpuscular hemoglobin 29 micromicrograms, mean corpuscular volume 87 cubic micra, mean corpuscular hemoglobin concentration 35%. White blood cell count 7,600/mm³ with 73% segmented neutrophils, 1% neutrophilic bands, 26% lymphocytes. The red cells were normochromic, normocytic and platelets were normal in number and morphology. Pregnancy test was negative. Electrolytes were within normal limits. Lactic dehydrogenase 363 IU/L (normal up to 270 IU/L). Alkaline phosphatase, transaminase, bilirubin, calcium, total protein, inorganic phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, and cholesterol were within normal limits. A chest film was unremarkable and excretory urogram revealed a minimal residual of urine in the bladder but no abnormalities of the kidneys, pelvis, or ureters, were noted.

DR. FOSTER: I will start with a brief differential diagnosis of testicular masses.

Syphilitic gumma is a great rarity in this era. A serologic test such as VDRL for syphilis might have helped although it could have been negative even in the presence of a gumma. A fluorescent treponema antibody test (FTA) would have been positive. Tuberculosis is another rare cause of a mass in the testis which we can exclude probably since there is no other clinical evidence for it.

Epididymitis is one of the most frequent conditions misinterpreted as a testicular tumor but the reverse situation in which testicular tumor is confused with the more common entity of epididymitis is also the case. Blandy has suggested that if there is no pyuria or bacteria in the urine and if the swelling is not clearly confined to the epididymis exploration must be carried out. Whitmore¹⁹ takes

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a more conservative approach and suggests vigorous treatment of a case thought to be clinically epididymitis, and then exploration if there is no clear evidence of improvement.

Another important problem requiring immediate attention is testicular torsion since testicular pain and swelling can suggest testicular torsion as well as epididymitis or testicular tumor. The management of all these entities often should begin with exploration.

I could mention, at this point, that the differential diagnosis between testicular torsion and epididymitis can be aided with the use of doppler to detect blood flow. In testicular torsion the blood flow is usually decreased whereas in epididymitis the blood flow is increased to the testis. However, Nasrallah¹⁸ has reported cases of increased blood flow resulting from hyperemia of the skin in cases of torsion. It has been suggested that in more painful lesions of testis, the doppler study is likely to be more helpful. This is because the patient seeks attention early before the increased blood flow to the scrotum can occur. This increased flow often will take place after 18 to 24 hours. The doppler test as advocated by Perri¹⁷ will probably only obviate exploration in a small percentage of cases.

A group of common cystic lesions may be difficult to differentiate from testicular neoplasm. These include hydrocele, hematocele, spermatocele, cyst of the epididymitis, and varicocele. A simple measure in detecting some of these lesions is transillumination of the mass. Certainly, hydroceles and spermatoceles will often transilluminate.

Ultrasound has been used as a modality to investigate the scrotal contents in suspected mass lesions. It will indicate the exact location of the lesion and determine whether it is cystic or solid. In one study evaluating ultrasonic findings, the final diagnosis in 27 patients (54 testis) indicated 25 normal testis were correctly identified, 14 abnormal extratesticular conditions were divided into 10 hydroceles, 4 spermatoceles, and 7 solid abnormal extratesticular abnormalities were due to epididymitis. Six testicular neoplasms of solid nature were correctly identified as to their solid nature and testicular location. A hemorrhagic hydrocele and a scrotal sarcoma gave indeterminate findings. The ultrasonic diagnosis was 96% accurate. The author has advised caution and suggests that ultrasound only be used in confirmation of the diagnosis of benign scrotal processes. If a suspicion of testicular tumor exists, surgical exploration through a high inguinal approach is mandatory.

Scrotal thermography and scintiscanning have yet to achieve full acceptance.

Even allowing for a history of trauma, hematocele and rupture of the testis may be difficult to separate from tumor preoperatively but exploration and inspection will settle the issue and offer a surgical remedy. Very occasionally a hernia may prove difficult to differentiate on physical examination. Ultrasonic study would demonstrate the extra-testicular nature of the lesion.

It has been a common teaching that pain is usually not associated with testicular neoplasms but this is incorrect. A study of 510 patients revealed that pain occurred in about half of the cases of testicular tumors.

Trauma has not been proved to be an etiologic factor in testicular neoplasm. It is felt that the enlargement of the testes caused by the neoplasm brings about a situation in which trauma is more likely. It is noted in the protocol that the testis had been descended since birth. This is quite a significant part of the history as it is well known that undescended testes have at least a 20 times more likely tendency to undergo malignant change. Testicular neoplasms are divided into the non-germ cell tumors which comprise only 4% of the total and the germ cell tumors which comprise the remaining 96%; the latter are all malignant.

Seminoma is the most common malignant germ cell neoplasm noted in undescended testes. Malescent does not cause an earlier occurrence of the tumor and orchidopexy has not lessened the subsequent risk of tumor formation in the previously undescended testis. This has been disputed and it has been claimed that if orchidopexy is done before the age of six years the incidence of malignancy is markedly reduced. However, early orchidopexy is a relatively new procedure and more time for evaluation of this technique is needed. However, no tumors have been noted in testes which have been brought into the scrotal cavity before six years of age.

The fact that the patient had no previous surgery on the testis is a significant point in the eventual management of this patient should the patient have a testicular tumor. If he had previously undergone some inguinal or scrotal procedure, this would have disrupted the lymphatic drainage of the testis and a different surgical approach would be required. It should be noted that the testis does not drain to the inguinal lymph nodes but lymphatic drainage occurs up into the paranephric retroperitoneal region. There is often a satellite lymph node which is situated lateral to these paranephric retroperitoneal lymph nodes. The appreciation of this satellite or sentinel node is an important point as pedal lymphangiography will not detect metastasis to the satellite

Table 1. Comparison of Classification of Testicular Germ Cell Tumors

Dixon and Moore (1952)	Collins and Pugh (1964)	British Testicular Tumour Panel (1976)	Mostofi and Price (1973)	W.H.O. (1976)
Seminoma	Seminoma- classic spermatocytic	Seminoma- classic spermatocytic	Seminoma- typical anaplastic spermatocytic	Seminoma- typical anaplastic spermatocytic
Embryonal carcinoma	Malignant teratoma anaplastic (MTA) Malignant teratoma, in- termediate, with no differentiated or organoid elements (MTIB)	Malignant teratoma, undifferentiated (MTU)	Embryonal carcinoma adult polyembryoma	Embryonal carcinoma
Teratoma with embryon- al carcinoma ("terato- carcinoma")	Malignant teratoma, in- termediate, with dif- ferentiated or organoid elements (MTIA)	Malignant teratoma, intermediate	Embryonal carcinoma with teratoma ("Teratocarcinoma")	Embryonal carcinoma with teratoma ("Teratocarcinoma")
Teratoma, adult	Teratoma, differentiated (TD)	Teratoma differentiated	Teratoma, mature	Teratoma, mature
Choriocarcinoma	Malignant teratoma, trophoblastic (MTT)	Malignant teratoma, trophoblastic	Choriocarcinoma	Choriocarcinoma
	Orchioblastoma	Yolk sac tumor	Embryonal carcinoma, infantile (juvenile)	Yolk sac tumor

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lymph node.

Previous surgery would have distorted markedly the lymphatics and made pedal lymphangiography very difficult. What is more, surgical trauma would have altered the lymphatic pathway so that the testicular neoplasm might drain into scrotal lymphatics and secondarily into inguinal lymph nodes.

Germ cell neoplasms of the testis are malignant and divided into seminomas which all arise from a primitive germ cell of the testis and the non-seminomatous germ cell neoplasms which include embryonal carcinoma, mature and immature teratomas and choriocarcinoma. This distinction has clinical implications since seminomas are radiosensitive while the nonseminomatous germ cell tumors are not.

Nomenclature has been a stumbling block to understanding of the various testicular germ cell tumors. I have included under Table 1 a comparison of the classification of the testicular germ cell tumors from an article by Nochomovitz¹ from a symposium in the **Urologic Clinics of North America** in October, 1977.

Before operating on a testicular tumor, several baseline studies should be obtained. These include a test for alpha fetoprotein (AFP) and human chorionic gonadotrophin (HCG). Both determinations for these tumor markers are done by radio-immunoassay and have significance in identifying the type of tissue present in the tumor, as well as in

monitoring both the extent of the tumor and in gauging the therapeutic success in treating these neoplasms. Lange³ in the October, 1977 **Urologic Clinics of North America** made the following points: 1) Seminomas never have elevated AFP. 2) Patients with seminoma and high HCG titers may not respond to the usual therapy as well as patients who do not have high HCG titers. 3) False positive marker levels of HCG and AFP indicating metastatic disease prior to lymphadenectomy have not occurred provided adequate time for clearance of the marker following orchiectomy has occurred. This means that after orchiectomy a high titer of either of the substances mentioned would indicate metastatic disease. Unfortunately false negative levels do occur. 4) All patients who have remained free of disease and in complete therapeutic remission on chemotherapy have had no detectable marker levels. Rising levels have predicted regression or progression of disease 3 to 6 months before other clinical indicators. 5) In regard to differential diagnosis when both tumor markers are negative, testicular masses are often benign. Rarely seminomas and mature teratomas can cause false negative determinations. The presence of elevation of either one of these tumor marker levels in a patient with a testicular swelling certainly suggests a germ cell tumor of the testis. 6) False negative values in nonseminomatous germ cell tumors which are metastatic are rare. It is strongly recommended that pa-

tients with clinical evidence of metastatic non-seminomatous germ cell disease and consistently negative markers should have a biopsy specimen taken before chemotherapy is begun or continued.

In summary, the most significant use of these tests is in monitoring therapy. Changing levels have reflected or predicted by 3 to 6 months progression or remission of disease. A patient with an enlarging mass in the retroperitoneum or elsewhere and negative levels often has pure seminoma or mature teratoma or a benign lesion. It should be noted that even though the original testicular neoplasm can be a lesion such as embryonal carcinoma, the metastases may be different. This is somewhat confusing but it has been documented on numerous occasions. These two markers, AFP and HCG, are more sensitive for the presence of metastases than any other diagnostic procedure including lymphangiogram, computer axial tomogram, or ultrasonic measurements. When they are positive they are strongly indicative of the presence of a germ cell tumor. However, false negatives do occur.

The initial surgical approach to the patient is the same regardless of tumor type. This should be through an inguinal skin crease incision. The cord should be clamped and the testis delivered from the scrotum through the incision. The organ can then be inspected and a decision made for further therapy. Should the testis be obviously ruptured or a torsion of the testis be present, this can be corrected and the testis returned to the scrotum. In questionable cases, the tunica vaginalis may be opened and the surface of the testis inspected. Testicular tumors which do not transilluminate should not be subjected to needle biopsy or approached through a scrotal incision for biopsy. This can lead to direct implantation of tumor in the incision and a distortion of the lymphatic drainage making the lesion virtually inoperable.

There are several methods for clinical staging of testicular tumors. The following one was developed in Great Britain and is as good as any.

- Stage I: Tumor confined to the testis. (Negative lymphangiogram and negative levels of HCG and AFP)
- Stage II: Positive lymphangiogram without disease above the diaphragm
- Stage III: Mediastinal and supraclavicular node disease is present
- Stage IV: Extralymphatic spread to visceral organs is present

Testicular neoplasms spread to lymphatics in the retroperitoneal space. Extension to the mediastinum and supraclavicular lymph nodes occurs subsequent-

ly. The lungs, kidney, pancreas, brain and bone may be involved by a hematogenous route. Seminomas usually will show lymph node metastases early but visceral spread by hemotogenous means is a late phenomenon. This, however, is not true of the non-seminomatous germ cell neoplasms which spread frequently via the blood stream and lymphatics early.

Now let us return to the particular patient under discussion. He has a good chance of having a pure seminoma. I base this on the fact that he has negative levels for HCG and AFP which are more commonly positive in the nonseminomatous germ cell tumors. We have no evidence of metastases to the lungs or retroperitoneum. Treatment would consist of radical orchiectomy followed by radiation to the nonaortic and iliac lymph nodes. If the patient had been thought to have mediastinal or supraclavicular lymph node areas involved as in stage II or III disease, these areas would also have to be irradiated. In patients with metastatic disease above or below the diaphragm, additional chemotherapy with an alkylating agent would be reasonable. The prognosis for stage I and early stage II seminoma is very good. Ninety to 95% are alive and free of disease at three years. Seventy percent of patients with retroperitoneal masses are alive and free of disease at three years and 30% to 60% of patients with metastatic disease to the lymph nodes above the diaphragm are free of disease at three years. It is to be noted that seminomas are extremely radiosensitive.

Should the patient have a nonseminomatous germ cell tumor, he should probably have a bilateral retroperitoneal node dissection, regardless of the markers, in addition to radical orchiectomy. If the patient had stage I disease, he has a better than 90% chance of cure. Adjunctive chemotherapy is not indicated in stage I disease for nonseminomatous germ cell tumors. These neoplasms should be followed by AFP and HCG determinations and any rise in these markers should be a cause for immediate reevaluation. Any patient who has stage II or stage III disease due to nonseminomatous germ cell tumors should have chemotherapy with one or more of the current regimens. Cisplatinum, vinblastine, and bleomycin are one drug combination which has met with good success. These patients have an overall chance of 75% for complete remission. Therapy with BCG maintenance has also been used for nonseminomatous germ cell tumors.

Before I close I would like to consider some problems posed by the therapy used. Many patients are interested in sperm preservation via cryoprotection before they undergo radiotherapy. It should be

noted that sperm motility after 6 to 12 months is markedly reduced and this technique of sperm preservation is very costly. Therefore, one could caution against false hopes until storage techniques are improved. The other question the patient may ask is what kind of sexual function he is going to have, particularly after a retroperitoneal lymphadenectomy. Nearly all patients report sexual dysfunction after lymphadenectomy, but nearly 95% will have an erection and dry orgasm with no ejaculation. An occasional patient will be able to produce semen. Retrograde ejaculation apparently does not occur.

Dr. Foster's Diagnosis
Malignant Germ Cell,
Neoplasm of Testis

*DR. LOREN AMUNDSON: Which is the most common tumor?

DR. FOSTER: Seminoma.

DR. BARLOW: We received a right testis with attached epididymis and a long segment of spermatic cord. This picture (Fig. 1) shows a 3 cm. nodule with focal hemorrhage. The presence of hemorrhage is unusual in seminoma and often denotes embryonal carcinoma or choriocarcinoma. On multiple sections pure embryonal cell carcinoma was found. This photomicrograph (Fig. 2) shows a characteristic tumor with irregular microcystic spaces and sheets of cells with large nuclei, prominent nucleoli, and abundant mitoses. Although the tumor did not extend through the tunica albuginea or involve the spermatic cord, vascular invasion of the epididymis was present.

FINAL DIAGNOSES
EMBRYONAL CELL CARCINOMA OF
THE TESTIS WITH INVASION OF THE
EPIDIDYMIS AND VASCULAR INVASION

DR. JOHN HOSKINS: In this case, there were classical findings on physical examination for testicular neoplasm. However, as has been pointed out by Dr. Foster, the AFP and HCG can be used in a differential diagnosis of testicular neoplasms. When these are elevated, a germ cell neoplasm is certain to be present. However, a negative test does not mean exploration is not necessary. I would like to repeat again that these lesions should not be approached through a scrotal incision. The lymphatics draining the testis to the retroperitoneum rarely have branches. When a scrotal incision is used instead of an inguinal incision, or if a needle biopsy of the mass is attempted, subsequent surgery neces-

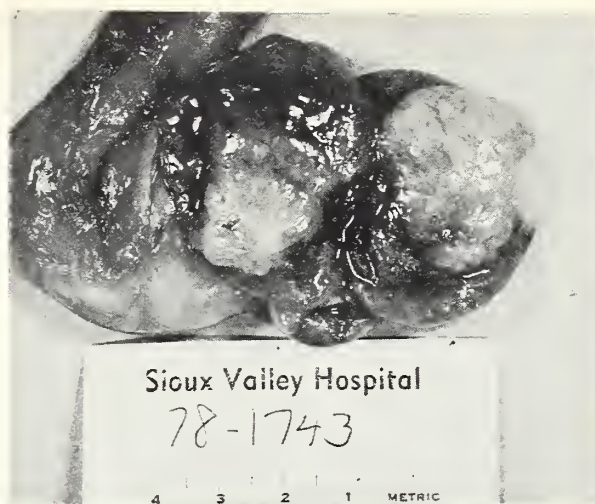


Figure 1
Cross section of embryonal carcinoma of testis. Dark areas represent hemorrhage.



Figure 2
Microscopic pseudoglandular pattern of embryonal carcinoma (100 x).

sitates excising the scar and involved scrotum and removing inguinal lymph nodes. The scar, involved scrotum, and inguinal lymph nodes must be removed en bloc. It should be noted that through the inguinal incision, the testis can be inspected and extratesticular tumor can be removed for frozen section. There may be less morbidity from the inguinal excision than from the scrotal incision. I have been involved in a study in which randomized patients, who were undergoing orchiectomy for prostatic cancer, were divided into a group having a scrotal incision and those in whom inguinal incisions were used for orchiectomy. The patients with the inguinal incision had less morbidity than those with the scrotal incisions.

Our next step was to perform retroperitoneal lymph node dissection.

*Professor and Chairman of Family and Community Medicine, School of Medicine, University of South Dakota.

There has been considerable argument as to whether to do unilateral or bilateral retroperitoneal lymph node resection. I chose the bilateral procedure. The patient had 5 of 26 lymph nodes which contained metastatic embryonal cell carcinoma. Since the patient was stage II and because of the cell type, chemotherapy was indicated. We are using a protocol in conjunction with Mayo Clinic which is composed of vinblastine, actinomycin D, and bleomycin for a 1½ month period. The patient is now being placed on maintenance therapy with vinblastine, actinomycin D, and methotrexate.

No one knows how long to use maintenance therapy. It used to be said that if a patient survived two years after one of these neoplasms, his chances for prolonged survival were excellent. However, with the use of chemotherapeutic agents, tumor growth may be suppressed and metastatic disease may appear after chemotherapy is discontinued beyond the two year period. Therefore, patients with testicular neoplasms should be continued to be followed for a long period of time.

The problem of infertility after radiation of the retroperitoneum has been brought into focus by a law suit against a physician. Immediately after radiation therapy for a seminoma, the patient became infertile. Studies regarding men involved in radiation accidents have suggested that fertility does return after five years.

The undescended testis is certainly predisposed to malignant testicular neoplasm. An intraabdominal testis can have up to 40 times the chance of developing neoplasm compared to the descended testis. In most instances, where orchidopexy was performed and neoplasm occurred, the orchidopexy was performed at puberty or after. The concept of doing orchidopexies before the age of 6, although not new, is certainly recommended. Recent information suggests that it prevents future testicular neoplasms. In fact, the earlier the orchidopexy is done, the less likely subsequent tumor will occur. A recent recommendation has been to do orchidopexy before two years of age.

The loss of ejaculation after retroperitoneal lymph node dissection is certainly a problem. It reflects loss of peristalsis of the ductus deferens and loss of contraction of the seminal vesicles. Sympathomimetic drugs are being evaluated to achieve ejaculation in this situation.

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*DR. LLOYD SWEENEY: If a testis is absent and it cannot be found in the inguinal canal, is this an indication to do an intraperitoneal exploration and bring the testis down?

DR. HOSKINS: Opinion is changing on this. I believe the testis should be sought and be brought down into the scrotum. There are some tricks for looking for the testis. The testis may be found on a short pedicle or mesentery within the peritoneal cavity rather than in the retroperitoneum. Most of the testes which are intraabdominal are found just inside the internal inguinal ring.

DR. SWEENEY: Then you try to bring the testis down rather than taking it out? What about the high incidence of malignant degeneration?

DR. HOSKINS: If the procedure is done early enough, there is not a high incidence of malignant degeneration. I should also mention that when a patient has an absent testis and abdominal pain, one must consider a torsion of an intraabdominal testis. It is interesting to note that most intraabdominal testes which undergo torsion have neoplasms.

**DR. JOHN OCHSNER: Although orchidopexies are being performed at an earlier age now and the incidence of malignant degeneration is not as high, I would like to note that physicians should try to warn their patients who have had orchidopexies after 6 that malignant degeneration is possible. Examination of the testes by a physician and perhaps more important self examination of the testis is mandatory in these patients. I would also like to note that tumors can occur in atrophic testes.

DR. BARLOW: Concerning testicular neoplasms, there has been much confusion because of their relative rarity, their great variety, and the fact that a variety of histologic patterns can be seen in the same tumor. When one considers that only 1% of male cancer deaths are due to testicular neoplasms, this sounds like a rather insignificant figure. However, 11% to 13% of all cancer deaths between the ages of 15 and 34 years in males are due to this neoplasm.¹² I like to segregate testicular neoplasms by age groups.

One tumor seen in children is a well differentiated or mature teratoma which is invariably benign. The other tumor has formerly been called embryonal carcinoma of infancy and is better called endodermal sinus tumor or yolk sac tumor and is the most common malignant testicular tumor of childhood. Sixty-five percent of tumors in children occur under the age of 2½ years. Eighty percent of these tumors are malignant.

In the young adult male, germ cell tumors are by far the most common. Seminoma is the most com-

mon tumor and most series estimate that approximately half the germ cell malignancies of the testis are due to this neoplasm. The neoplasm is highly radiosensitive and survival is excellent if the neoplasm is pure. Spermatocytic seminoma has a reasonably good prognosis but is rare. There is another variety called atypical seminoma and this has a poor prognosis but fortunately occupies only a small percentage of seminomatous neoplasms.

The most common nonseminomatous germ cell is the embryonal carcinoma. Survival rate although improving with chemotherapy has in the past been only 35% for three years. Our patient today falls in this category. Teratomas of the testis occupy 5% to 10% of the germ cell neoplasms and have an excellent prognosis if they are mature, and somewhat worse prognosis if they contain immature elements. It should be pointed out, however, that mature teratomas in adults, unlike those found in infants, do metastasize when they occur in the adult patient. The rare choriocarcinoma of the adult testis has a dismal prognosis since they do not respond to chemotherapy. This is in contrast to the lesion seen in the female uterus. As a generalization, it can be said that in mixed neoplasms, one or more of the above histologic patterns may be found. The more seminomatous tissue that is present, the better the prognosis. The presence of any elements other than seminoma markedly reduces the prognosis. The presence of choriocarcinoma markedly impairs survival and the presence of embryonal carcinoma also tends to reduce survival patterns.

Germ cell neoplasms after the age of 40 are rare. The tumor most often seen in the elderly male after 50 or 60 years of age is the histiocytic lymphoma of the testis. This has a notoriously poor prognosis.

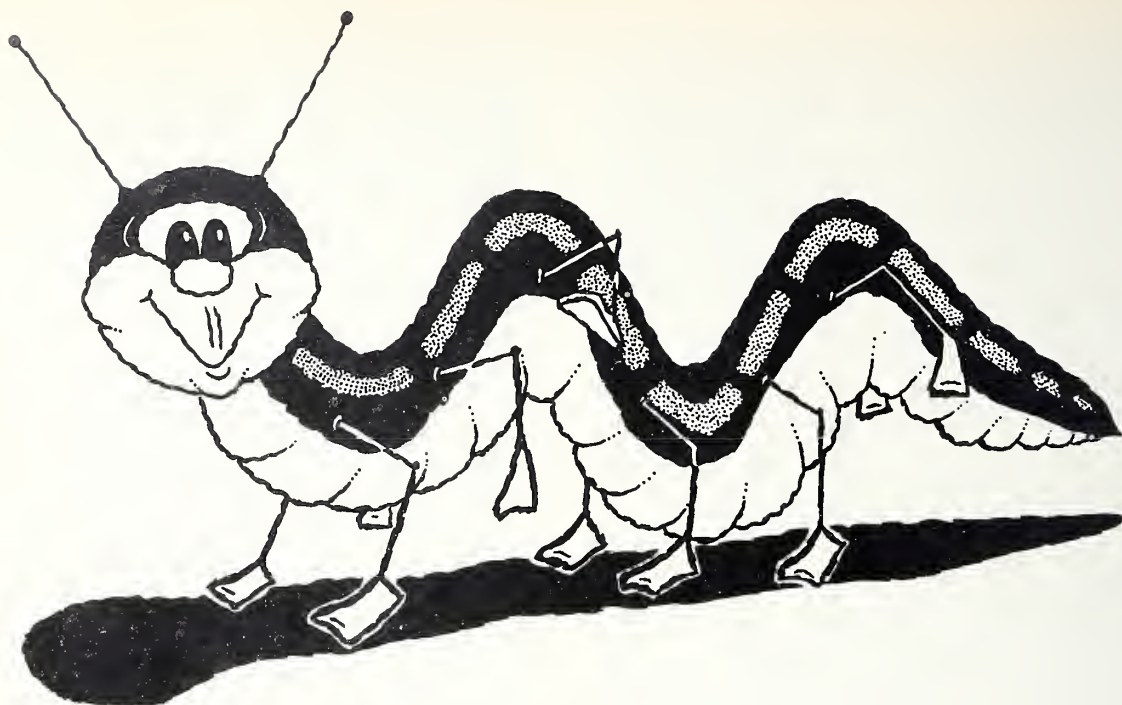
In closing, I would like to say a few words about the tumor markers. AFP is a glycoprotein that is found in the fetal yolk sac, liver, and gastrointestinal tract. It is a major serum protein of the fetus but the level decreases toward term and then decreases markedly after birth until it is barely detectable by radioimmunoassay at one year of age. The measurement of the compound first became popular in detecting hepatocellular carcinoma. Later it was found to be elevated in endodermal sinus tumor of the infant testis and embryonal carcinoma of the young adult. Occasionally, AFP may be elevated in pancreatic or gastric carcinoma and rarely in renal cell carcinoma, transitional cell carcinoma or prostatic carcinoma. It is not elevated in seminoma of the testis as Dr. Foster has pointed out.

HCG is a placental hormone. The measurement of this hormone can be made specific by using the

so-called beta subunit assay. The glycoprotein hormones such as luteotrophic hormone (LH) and HCG both have alpha and beta chains. The alpha chains are identical but the beta chains are specific for each compound. Therefore, if an antibody is developed to the beta chains, one has a specific assay. HCG has been the classic test for monitoring hydatidiform mole and choriocarcinoma in the female. However, it is clearly elevated in nonseminomatous germ cell tumors as well as in some seminomas. It can also be elevated in pancreatic, gastric, colonic and pulmonary carcinoma. The fact that the presence of HCG levels in the serum of a patient with pure seminoma indicates a poor response has been mentioned by Dr. Foster. At any rate, it is clear that both of the above tumor markers can be used as serial monitors in the therapy of testicular neoplasms and maybe an aid in the differential diagnosis of scrotal masses.

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ointment may be used to prevent bacterial contamination in burns, skin grafts, incisions, and other clean lesions. For abrasions, minor cuts and wounds accidentally incurred, its use may prevent the development of infection and permit wound healing.

CONTRAINDICATIONS: This product is contraindicated in those individuals who have shown hypersensitivity to any of its components. Do not use in the eyes or in the external ear canal if the eardrum is perforated.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

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secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

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we all raced down to Sam.
He stood there hugging
a proud Mrs. Wattles and
beaming at us all.*



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Sam Higgins would go out of his way to wiggle his ears at a baby. Or do his magic act for the older children. It was easy to see the reason. Kids loved Sam 'cause they knew he loved them.

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Future Meetings

April

American Occupational Health Conference, Disneyland Hotel, Anaheim, CA, April 30-May 4. Fee: \$45—AOMA members; \$65—non members. AMA Category I credits. Contact: AOMA Headquarters Office, 150 North Wacker Dr., Chicago, IL 60606.

May

A Symposium on Structure-Function Relationships in Proteins, Nucleic Acids and Viruses, U. of Minn., Minneapolis, MN, May 2-4. Contact: Office of CME, Box 293 Mayo Memorial Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Pain-Live Clinic, V. A. Hospital, Fort Meade, SD, May 3. 1 hr. AMA Category I credit. Contact: Office of Cont. Health Ed., USD, Sioux Falls, SD 57105, (605) 339-7573.

Pain-Live Clinic, V. A. Hospital, Hot Springs, SD, May 4. 1 hr. AMA Category I credit. Contact: Office of Cont. Health Ed., USD, Sioux Falls, SD 57105, (605) 339-7573.

Management of Colorectal Problems, Sheraton-Ritz Hotel, Minneapolis, MN, May 4-5. Fee: \$85. AMA Category I credits. Contact: Office of CME, Box 293, Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

South Dakota Society of American Association of Medical Assistants Annual Convention, Gill's Sun Inn, Rapid City, SD, May 4-6. Contact: Ann Whetson, 2311 Smith Ave., Rapid City, SD 57701.

Ninth Annual Meeting of the Great Plains Organization for Perinatal Health Care, Radisson South Hotel, Bloomington, MN, May 10-12. Contact: Mrs. Virginia Rittenour, Coord., Great Plains Organization, Box 50, 420 Delaware St., S.E., Minneapolis, MN 55455.

EKG Interpretation and Arrhythmia Management, Water Tower Hyatt, Chicago, IL, May 11-13. Fee: \$202. 15 hrs AMA Category I and AAFP credits. Contact: International Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Cardiology Today, U. of Iowa, Iowa City, IA, May 14-17. AMA Category I and AAFP credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Marriage and Divorce Counseling, V. A. Hospital, Fort Meade, SD, May 17. 1 hr. AMA Category I credit. Contact: Office of Cont. Health Ed., USD, Sioux Falls, SD 57105, (605) 339-7573.

Comprehensive Care of the Child with a Disability: New Concepts, Minneapolis Children's Health Center, Minneapolis, MN, May 18-20. Fee: \$60. 16 hrs. AMA Category I and AAFP credits. Contact: Joseph Capell, M.D., Children's Health Center, 2525 Chicago Ave., S., Minneapolis, MN 55404.

Pain: Diagnosis and Treatment of the Difficult Patient, McKennan Hospital, Sioux Falls, SD, May 19. 8 hrs. AMA Category I credits. Contact: Bill Arbes, Ph.D., Dept. of Psychiatry, USD School of Medicine, Sioux Falls, SD 57105, (605) 339-6785.

14th Annual Meeting of the Association for the Advancement of Medical Instrumentation, Caesars Palace, Las Vegas, NV, May 20-24. AMA Category I credits. Contact: AAMI, 1901 N. Fort Myer Dr., Suite 602, Arlington, VA 22209.

Family Practice Review: Update 1979, Radisson St. Paul Hotel, St. Paul, MN, May 21-25. Fee: \$275. AMA Category I credits. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Current Concepts in Radiation Therapy, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 23-25. Fee: \$200. AMA Category I credits. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Laboratory Medicine Update 1979, IDS Center Conf. Theatre, Minneapolis, MN, May 23-25. Fee: \$200. AMA Category I credits. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Gastric Bypass, U. of Iowa, Iowa City, IA, May 24-25. AMA Category I and AAFP credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Practical Dermatology in Primary Care, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 31-June 2. Fee: \$150. AMA Category I credits. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

June

Clinical Hypnosis, Earle Brown Con. Ed. Center, St. Paul, MN, June 1-2. Fee: \$150. AMA Category I credits. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Contemporary Therapy of Cardiovascular Disease, Cragun's, Brainerd, MN, June 1-3. Fee: \$175 - members of AHA, \$225 - non members. AMA Category I credits. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Asthma, New York Sheraton, New York City, June 6-8. Fee: \$155 - American College of Chest Physician members, \$180 - non members. 15 hrs. AMA Category I credits. Contact: Dale E. Braddy, Dir. of Ed., ACCP, 911 Busse Highway, Park Ridge, IL 60068.

Workshop on Prevention of Coronary Heart Disease, Spring Hill Center, Wayzata, MN, June 6-8. Fee: \$225. AMA Category I credits. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

South Dakota State Medical Association Annual Meeting, Howard Johnson Motor Lodge, Rapid City, SD, June 8-10. Contact: SDSMA, 608 West Ave., North, Sioux Falls, SD 57104.

Management of Cerebral Palsy, Children's Rehabilitation Center, U. of Minn., Minneapolis, MN, June 11-16. Fee: \$235. **Optional Practicum**, June 18-22. Fee: \$150. AMA Category I credits. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Hepatic, Biliary and Pancreatic Surgery, West Bank Aud., U. of Minn., Minneapolis, MN, June 13-16. Fee: \$250. AMA Category I credits. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

(continued)

Advanced Workshop: Sexual Issues in Psychotherapy, U. of Minn., Minneapolis, MN, June 15-16. Fee: \$75. AMA Category I credits. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Special Course on Alcohol and Drug Abuse Treatment, U. of Utah Hosp. Med. Center, Salt Lake City, UT, June 18-21. Fee: \$200. 25 hrs. AMA Category I credits. Contact: James R. Swenson, M.D., P.O. Box 2604, Salt Lake City, UT 84110.

Scientific Seminar on Advances in Pediatrics, Sylvan Lake, Custer, SD, June 20-22. AMA Category I and AAFP credits. Contact: Thomas Aceto, Jr., M.D., Dept. Med., McKennan Hosp., Sioux of Pediatrics, U. of S.D. School of Falls, SD 57101.

Natural Family Planning Seminar, Marriott Inn, Bloomington, MN, June 21-22. Fee: \$100. AMA Category I credits. Contact: Office of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

July

Allergy and Immunology, Colby College, Waterville, ME, July 2-5. 16 hrs. AMA Category I credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby Coll., Waterville, ME 04901.

Joint Meeting of American Society of Clinical Pathologists and College of American Pathologists, Washington, DC, July 7-14. Contact: American Society of Clinical Pathologists, 2100 W. Harrison St., Chicago, IL 60612.

Topics in Emergency Medicine, Colby College, Waterville, ME, July 8-12. 24 hrs. AMA Category I credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby Coll., Waterville, ME 04901.

Epilepsy Course, Colby College, Waterville, ME, July 10-14. 18 hrs. AMA Category I credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby Coll., Waterville, ME 04901.

Topics in Pediatrics, Colby College, Waterville, ME, July 15-19. 17 hrs. AMA Category I credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby Coll., Waterville, ME 04901.

Surgical Techniques, Colby College, Waterville, ME, July 17-20. 15 hrs. AMA Category I credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby Coll., Waterville, ME 04901.

Hematology, Colby College, Waterville, ME, July 22-26. 22 hrs. AMA Category I credits. Contact: R. H. Kany, dir., Div. of Special Pro-

grams, Colby Coll., Waterville, ME 04901.

Neurosurgery, Colby College, Waterville, ME, July 22-26. 21 hrs. AMA Category I credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby Coll., Waterville, ME 04901.

Summer Institute of Alcohol Studies, Duke U., Durham, NC, July 22-27. AMA Category I credits. Contact: Fritz Anlyan, Summer Ed. Programs, Duke U., 06B West Duke Bldg., Durham, NC 27708.

Coronary Disease, Exercise Testing, and Cardiac Rehabilitation, Playboy Resort, Lake Geneva, WI, July 27-29. Fee: \$202. 13 hrs. AMA Category I credits. Contact: International Med., Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Dermatology, Colby College, Waterville, ME, July 29-Aug. 2. 16 hrs. AMA Category I credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby Coll., Waterville, ME 04901.

F. T. Hill Otolaryngology Seminar, Colby College, Waterville, ME, July 29-Aug. 2. 18 hrs. AMA Category I credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby Coll., Waterville, ME, 04901.

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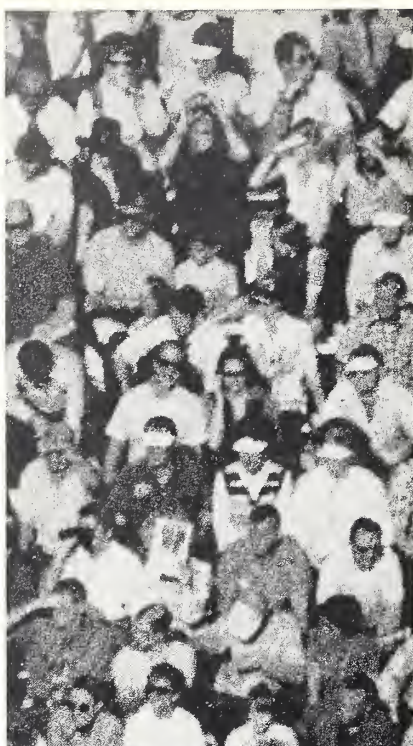
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SCIENTIFIC ARTICLES

- 9** Clinicopathological Conference
Seventy-Three Year Old Woman With Recurrent
Cystitis And Pneumaturia
Roger Olsen, M.D.
John F. Barlow, M.D.
- 31** Intramural Hematoma Of The Duodenum
A Case Report And Literature Review
Bruce C. Lushbough, M.D., D.A.B.F.P.
Saul Friefeld, M.D., F.A.C.R.
Joseph Primrose, M.D., F.A.C.S.

FEATURES

- 19** President's Page
- 20** Letter To The Editor
- 23** South Dakota AFP Chapter News
- 30** Laboratory Aids
South Dakota Pathology Page
- 37** Auxiliary News
- 39** Future Meetings



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INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence:* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy:* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children:* Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdose. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular:* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System:* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache, rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal:* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic:* Urticaria, rash, ecchymosis, erythema. *Endocrine:* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System:* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous:* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in mid-evening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in mid-morning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdose include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phenolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdose.

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Merrell

Seventy-Three Year Old Woman With Recurrent Cystitis And Pneumaturia

Roger Olsen, M.D.*
Discusser

John F. Barlow, M.D.**
Pathologist-Editor

Case No. M777 969

This 73-year-old caucasian female was admitted to Sioux Valley Hospital with the chief complaint of bladder pain, dysuria, frequency and passage of brown material and air on voiding.

One year prior to admission the patient had had dysuria, frequency, bladder pain, chills, fever and was evaluated at another hospital where a cystoscopy and intravenous pyelography were negative but a barium enema showed diverticulosis with associated spasm. An upper gastrointestinal series showed a hiatal hernia and a gallbladder series was within normal limit. Urine culture grew *Escherichia coli*; hemoglobin 8.6 gms/dl; hematocrit 27 vol.%, serum iron 61 mcgs/dl, iron binding capacity 412 mcgs/dl and a total iron saturation of 15% (consistent with borderline iron deficiency). Creatinine was 1.5 mgs/dl. Electrocardiogram showed high voltage in the limb leads suggesting left ventricular hypertrophy. The patient was discharged on iron therapy and did extremely well for eight months, but then began to develop recurrent urinary tract infection.

Review of systems was unremarkable. Past medical history revealed that she had had severe post partum hemorrhage with one of her children.

PHYSICAL EXAMINATION: Temperature 97.8°F., pulse 80/minute and regular, respirations 16/min. and regular, blood pressure 110 systolic and 80 diastolic. Examination of the head, eyes, ears, nose and throat were negative. The patient had marked kyphosis but the lungs were clear to auscultation and percussion. The heart rhythm was regular and the heart was not enlarged; there were no murmurs or extra sounds. Examination of the abdomen revealed no palpable organs or masses. On deep pressure, the sigmoid colon region was barely palpable and somewhat tender. There was minimal bladder tenderness. Neurologic examination was within normal limits.

LABORATORY DATA: Urinalysis—slightly cloudy, yellow;

specific gravity 1.020, pH 5.5, protein 3+ negative for glucose, ketone bodies, bile; large amount of hemoglobin; sediment 100-150 white cells/hpf, packed field of red cells, no casts. Prothrombin time 13 seconds with a 13 second control, partial thromboplastin time 31 seconds with a 26 second control. Hemoglobin 13.0 gms/dl, hematocrit 30 vol/dl and red count 4.35 million/mm³, mean corpuscular hemoglobin 30 micromicrograms, mean corpuscular volume 89 cubic micra, mean corpuscular hemoglobin concentration 32%, total leukocyte count 9,400/mm³, with 69% segmented neutrophils, 2% neutrophilic bands, 4% eosinophils, 23% lymphocytes, and 2% monocytes. The red cells were normochromic, normocytic, and the platelets were normal in number and morphology. A 12 panel test was within normal limits. pH 7.42, PCO₂ 49 torr, CO₂ content 32 mM/L, sodium 139 meq/L, potassium 3.4 meq/L and chloride 105 meq/L. Lactic dehydrogenase, alkaline phosphatase, aspartate aminotransferase (SGOT), total bilirubin, calcium, total protein, inorganic phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, and cholesterol were within normal limits. Several potassium determinations were low during admission with the lowest being 2.6 meq/L. A urine culture revealed over 100,000/ml of *Escherichia coli* resistant to ampicillin, carbenicillin, and nalidixic acid and sensitive to cephalothin, chloramphenicol, colistin, gentamicin, kanamycin, tetracycline, nitrofurantoin, sulfa, and sulfa-trimethoprim. An electrocardiogram was read as being within normal limits. X-ray examination showed a negative chest film. A cystogram and barium enema indicated extensive diverticulosis of the sigmoid colon and lower descending colon with associated diverticulitis and fistula formation extending from the sigmoid to the bladder margin. There was a probable abscess adjacent to the bladder as indicated by an indentation along the left lateral superior aspect of the bladder. A proctoscopy revealed fullness in the pelvis anteriorly. This was quite tender. The scope was inserted to 9 cm and the rectal wall appeared edematous. The examination could not be carried on after this because of patient discomfort. A cystoscopy revealed edematous papillary appearing bladder mucosa to the left of the midline on the posterior wall. There was no gas. A fistula could not be demonstrated. The area had the possible appearance of carcinoma but biopsies revealed acute and chronic inflammation. An operation was performed.

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*DR. L. LARSON: A barium enema, cystogram, and intravenous pyelogram were performed on the same day on this patient. In the cystogram, there is an indentation in the left superior aspect of the urinary bladder with irregularity of the mucosa and a suggestion of a soft tissue density around the indentation. (Fig. 1) The intravenous pyelogram showed a similar impression on the bladder in the same area. On colon examination, performed after the intravenous pyelogram, there is narrowing of the sigmoid colon and multiple diverticula which have a pointed appearance indicative of diverticulitis. One can also see contrast material extending from the colon inferiorly toward the bladder margin indicating a fistula between the colon and bladder. (Fig. 2) There is also a soft tissue mass surrounding the area consistent with an abscess.

DR. OLSEN: I feel this case represents a complication of chronic colonic diverticular disease and I would like to discuss that entity in general terms. It has been estimated that one-third of patients over the age of thirty-five and three-fifths of the patients over the age of seventy have colonic diverticular disease. When one considers there will be 24 million people in the United States 70 years old or older in 1980, then one can see that the economic loss as well as morbidity and fatality from this disease will be significant. Diverticular disease involves primarily the sigmoid and descending colon although it is pos-

sible to have solitary diverticula in the cecum and ascending colon but these are usually congenital in nature.

The muscular layer of the colon is composed of an inner circular layer and an outer longitudinal layer which coalesces into the taenia coli. There are weak points in the colonic wall between the taeni coli where the marginal branches of the colonic arteries penetrate the circular muscle of the colonic wall. These are favorite sites for herniation of the mucosa through the muscular wall. The proximity of the diverticula to the penetrating blood vessels explains the massive bleeding sometimes associated with diverticular disease.

It used to be thought that the complication of diverticulitis was due to obstruction of the narrow neck of the diverticulum with secondary inflammation and perforation of the diverticulum (similar to appendicitis). However, there has been no pathologic evidence that this is what actually occurs. Instead there is perforation of the fundus of the diverticulum with surrounding peridiverticular and pericolic inflammation. It is interesting that one third of the patients, operated on for painful diverticulitis, showed no evidence of inflammation but did show a thickened muscular wall without inflammation. This is referred to as painful diverticular disease. The pathogenesis of diverticular disease is due to the fact that patients with this entity have higher pressure

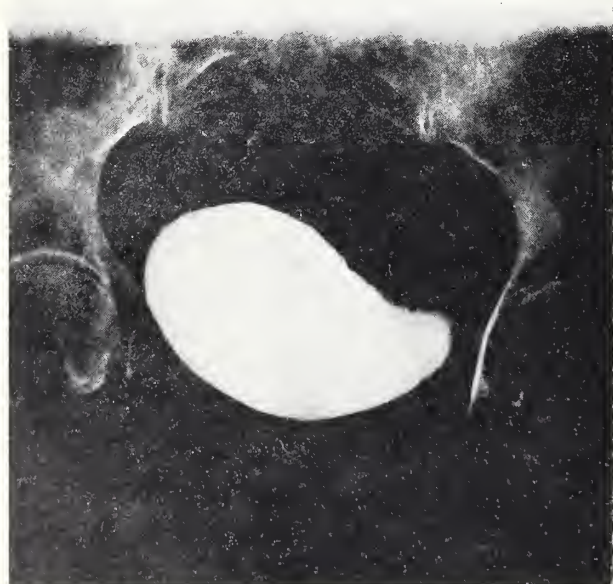


Figure 1

Indentation left superior aspect of bladder due to pericolic abscess.



Figure 2

Arrows show fistulous tract between bladder (below) and sigmoid colon (above). Diverticula can be seen in colon also.

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responses in the left colon to certain stimuli compared to normal people. Patients with diverticular disease had levels of pressure over 30 cm of water six times as often as other patients. The pressure elevations were produced by meals or other stimuli. For instance, it was found that morphine causes very high pressures in the sigmoid colon of patients with diverticular disease, but meperidine did not. For this reason, morphine should not be used as an analgesic in patients with diverticular disease.

When one considers LaPlace's Law, which states that the smaller the diameter of the sphere the greater the tension on the wall and the fact that the descending colon and sigmoid have the smallest lumen in the colon, then one can understand why there is high pressure within the lumen in the sigmoid. In addition, the sigmoid colon also undergoes segmentation into little bladder-like compartments in which there are very high pressures. This segmentation is necessary for retention of feces and for water absorption. The tremendous pressures in the small segments can force the mucosa through the weak points where vessels enter the muscular layer and diverticula are produced.

It often has been stated that diverticular disease is rare in Asia and Africa and more common in more advanced or civilized societies. One explanation has been the low fiber content of diets in civilized countries, but one cannot be sure of this. It is also true that in civilized society flatus retention is expected. This subjects the sigmoid colon to higher pressures for longer periods of time. Hostility and resentment can also cause increased sigmoidal pressures.

Diverticular disease should be suspected in patients over the age of 40 with lower abdominal pain, constipation, diarrhea or both and increased flatus. Diagnosis is best made by barium enema.

Diverticulitis is suspected when the patient has more severe or persistent pain localizing to the left lower quadrant and frequently extending into the lower back. This is also accompanied by anorexia, nausea, vomiting, or abdominal distention, significant ileus, or partial colonic obstruction. The patient may have diarrhea or constipation, tenesmus, fever, chills, polymorphonuclear leukocytosis and an elevated sedimentation rate. The patient may have increased urinary frequency or dysuria from involvement of the bladder or left ureter. One may find a large, tender, sausage-like fixed mass in the left lower quadrant on abdominal examination, but involuntary guarding may make it impossible to feel the mass. Rectal examination usually reveals a tender mass. If the patient has severe local or systemic signs of inflammation then the proctoscopic exami-

nation should not be done.

It can be very difficult to distinguish diverticular disease from irritable colon syndrome. The patient with either syndrome may develop flatulence, colonic pain, diarrhea, constipation, or abdominal pain, mucus in the stool, tenesmus, and occasionally bright red blood in the stool.

It also must be remembered that patients with diverticular disease are usually in an older age group and 20% of these patients may have an associated carcinoma of the colon or polyp in the sigmoid. The differential diagnostic points between these two common conditions of diverticulosis and carcinoma include a history of previous attacks of diverticular disease as opposed to no such history in carcinoma. Pain is more frequently severe with carcinoma and constipation more common and progressive in carcinoma than in diverticular disease. Massive hematochezia is more common in diverticulosis but anemia is more common in carcinoma. If there is a palpable mass, it is more likely to be nontender in carcinoma and tender in diverticulosis. Weight loss is common in carcinoma and the patients often have complete obstruction whereas in diverticular disease the patient is often obese and obstruction is rarely complete. Fever and leukocytosis are more common in diverticulosis. It may be very difficult to distinguish the narrowing of the sigmoid colon due to diverticulosis from carcinoma by barium enema. One suggestion has been to treat the patient for two weeks for diverticulosis to see if there is improvement. Glucagon may relax the spasm of diverticulosis but should not in carcinoma. It is important, however, to make the diagnosis of carcinoma in patients with diverticulosis early since the carcinoma which is associated with diverticulitis (incidence as high as 20%) has a five year survival rate of only 39% as compared to 50% five year survival in patients who have carcinoma of the colon without diverticular disease.

As mentioned previously diverticular disease occurs in approximately 60% of patients over 70 years of age. Approximately 25% of these will develop diverticulitis. Fifteen percent of these patients with diverticulitis will develop serious complications. This is particularly true if the patient has recurrent diverticulitis which will develop in 45% of patients who have diverticulitis. The major complications of recurrent diverticulitis are complete or incomplete colonic obstruction, hemorrhage and perforation which only rarely is free into the peritoneal cavity. Most common is the forme fruste or localized perforation into contiguous structures with abscess. The abscess may be complicated by gram negative septicemia or pylephlebitis. Fistulas may form with other

areas of the bowel, the vagina, the skin or most commonly the bladder.

This case represents a colovesical fistula. Diverticulitis is the most common single etiology for colovesical fistula. Approximately 50% of colovesical fistulas are secondary to diverticulosis with a reported range of 36-85%. One study showed 23% of patients who required surgery for diverticular disease had a colovesical fistula. They reported an overall incidence of colovesical fistulas in diverticulitis as 2-4%. The incidence of colovesical fistula is higher in males than females with a reported ratio of between 3-5:1. This is attributed to the uterus and adnexa separating the sigmoid colon from the bladder in the female. The incidence of colovesical fistula is apparently higher in women who have had a prior hysterectomy. However, it must be remembered that the incidence of diverticulitis is 1.5 to 3 times higher in males than in females. In females vaginal-colonic fistulas are seven times more common than colovesical fistulas.

The diagnosis of a colovesical fistula is often delayed for months because of slow progression of the disease. Early in the course an inflamed diverticulum probably becomes adherent to the bladder wall and eventually the inflammation erodes through the bladder musculature. During this time there may be recurring episodes of cystitis. Once the fistula is established, edema around the orifice into the bladder may be responsible for intermittent episodes of cystitis and also for difficulty in demonstrating the fistula track by radiography.

Colovesical fistulas may result from neoplastic disease or trauma. In a study at the Massachusetts General Hospital of 39 patients with colovesical fistula, inflammatory disease accounted for 27 (69%) of the cases and 24 of these patients had diverticulitis while two had ulcerative colitis and one had an appendiceal abscess. Other cases were carcinoma of the colon, carcinoma of the cervix, prostatic abscess, trauma, foreign body and unknown causes.

A wide range of symptoms is seen in patients with colovesical fistulas. Most patients have urologic symptoms often without bowel symptoms. This may delay the diagnosis of fistula secondary to inflammatory bowel disease. The most common symptoms are fecaluria, recurrent urinary tract infections, pneumaturia, hematuria, and recurring chills and fever. Some patients actually pass food particles through the urethra. The bowel symptoms include left lower quadrant abdominal pain, suprapubic pain, and loose bowel movements. It is unusual to pass urine rectally because sigmoid intraluminal pressure is generally much higher than vesical pressure but it is possible particularly with some bladder outlet ob-

struction and in patients with a prior colostomy and decreased sigmoid pressure. The previously mentioned study of 39 patients at the Massachusetts General Hospital showed that 54% of the patients had pneumaturia, 51% had fecaluria, 44% had symptoms of urinary tract infections, 41% of the patients had fever and chills, and 51% had hematuria. Only 5% of the patients passed urine by rectum. Pneumaturia may also occur in diabetic patients secondary to fermentation of urinary sugar by gas forming organisms such as *E. coli*. Fecaluria may be difficult to detect. One method is to have the patient void into a diaper so the brown color may be seen.

The demonstration of an enterovesical fistula is difficult but one of the most effective methods of demonstration is the use of visual dyes either given orally or through a colostomy. This is an inexpensive method. Agents used include charcoal, methylene blue, congo red, indigo carmine, and chromium sesquioxide. Excretory urography seldom confirms the diagnosis by demonstrating contrast material passing from the colon into the fistula tract. The fistula rarely involves the upper urinary tract. However, one of the more common fistula sites is the periureteral region in the bladder. Since these patients often require colonic surgery for treatment, intravenous pyelogram is recommended in patient evaluation.

Cystoscopy is the most important diagnostic study. The actual fistulous opening is seen less than half the time due usually to edema and inflammation about the orifice. Following barium enema, barium is sometimes seen in the bladder. Occasionally a methylene blue enema given just prior to the cystoscopy reveals dye seeping through a pinpoint opening into the bladder. Some reports reveal that suprapubic pressure at the time of cystoscopy may be helpful forcing colonic gas bubbles through the fistula but this is rarely actually present. It is important to do a bimanual pelvic examination following cystoscopy in an attempt to feel a left-sided pelvic mass. It is also important to perform biopsies when an occult fistula is suspected. Conversion of an occult fistula into an overt fistula is not serious and deep seated carcinoma in this region must be ruled out. It must be remembered that a normal cystoscopy does not rule out a colovesical fistula.

Cystograms may show some irregularity of the bladder mucosa or air in the bladder, but the edema around the orifice prevents the fistula from being seen one-third of the time. The barium enema has about a 30-40% success rate in demonstrating a colovesical fistula but it almost always shows diverticula, a mass, or obstruction proving it to be a valu-

able study. The cystoscopy after the barium enema also may reveal barium in the bladder. The absence of diverticula on barium enema examination does not rule out an occult fistula of diverticular origin. There can be a solitary diverticulum usually in the cecum or ascending colon of congenital origin producing the occult fistula. Occasionally a foreign body such as a chicken bone can be responsible for a fistula.

Sigmoidoscopy is seldom of any value in the diagnosis of colovesical fistula. The examination often reveals narrowing of the lumen, extrinsic compression, but pain or fixation of the colon due to angulation make the examination difficult. The sigmoidoscopic examination is necessary, however, to exclude carcinoma of the rectosigmoid region which is present in up to 20% of these patients, as well as ruling out ulcerative colitis or Crohn's disease.

Medical treatment of diverticular disease varies considerably depending upon the presence or absence of complications. Asymptomatic or mildly symptomatic diverticular disease requires no particular treatment but patients should be aware of their disease and should be told to notify their physician when they have a change in bowel habits, the passage of mucus or blood in the stool, fever, chills, urinary symptoms or abdominal pain. Early detection of diverticulitis and prevention of complications should be therefore accomplished.

No regimen has been shown to definitely decrease the incidence of complications or to alter the course of diverticular disease. However, the subjective response of 75-80% of the patients has been reported with high fiber diet in symptomatic diverticulosis. Increased bulk in the stool can be accomplished by hydrophilic colloid, fruits, and vegetables with fiber content or unrefined wheat. Patients should avoid foods with undigestible particles, seeds, popcorn, and nuts. Harsh laxatives and high edemas should be avoided while milder laxatives such as prune juice, magnesium sulfate, or mineral oil are usually effective. The anticholinergic drugs may decrease spasm and hypermotility but may very likely cause constipation. Their use is controversial.

Mild attacks of acute diverticulitis may be treated at home with bedrest, liquids or semiliquid diets and mild pain relievers. Anticholinergics may possibly be used. When fever, severe pain, leukocytosis and elevated erythrocyte sedimentation rate occur, the patient should be hospitalized on bedrest. Nasogastric suction may be necessary to put the bowel at rest and the patient should be given intravenous fluids and meperidine but not morphine. Blood cultures should be taken to rule out bacteremia which

is frequently due to bacteroides. If the cultures are positive, parenteral broad spectrum antibiotics such as ampicillin or a cephalosporin should be initiated. Occasionally an aminoglycoside must be added. Non-absorbable oral sulfonamides are of little value since the inflammation is extracolonic.

Colovesical fistulas rarely if ever heal spontaneously so surgery is indicated whenever the diagnosis is made. The single most important consideration is the healing of the anastomotic site of the colon resection. In a study by Rodkey and Welch the average length of colon excised in the patient who developed an anastomotic leak was 17.7 cms while the average length of colon was 25.4 cms in patients whose anastomosis remained intact. This indicates the need to resect a long segment of colon to provide soft flexible bowel at either end of the anastomosis. It has been suggested that neostigmine given to reverse the action of the muscle relaxants after anesthesia can cause a sharp contraction of the bowel tearing the anastomosis.

Surgical management remains controversial. The decision to do a primary resection and reanastomosis or staged procedure is difficult. Factors to be considered in choosing the operation are: age, general condition of the patient, toxicity, amount of bowel obstruction, extent of the fistula, presence of pericolic abscess, whether the ends of the anastomosis are free of diverticular disease and inflammation, and the preoperative preparation of the bowel. The classical staged procedure consists of: 1) transverse colostomy followed by a careful study to rule out carcinoma, 2) resection of the diseased colon with anastomosis of the normal bowel ends, and closure of the fistula, 3) closure of the previous colostomy stoma. This three stage procedure has stood the test of time and is associated with the best results. The major disadvantage is the protracted course. The average hospital stay for those with a primary resection and reanastomosis was only 17 days, while the average stay for a patient with a staged procedure was 53 days over a period of 3-5 months. The duration between the stages has become shorter with the use of broad spectrum antibiotics. The primary resection and reanastomosis now can be a safe procedure in selected cases.

In the patient presented today, the presence of fecaluria was diagnostic of some type of enterovesical fistula. The palpable tender mass in the sigmoid area was certainly compatible with diverticulitis. A history of recurrent urinary tract infection was certainly consistent with enterovesical fistula. We have no evidence that the patient had Crohn's disease or ulcerative colitis to produce the fistula.

Dr. Olsen's Diagnosis

Diverticular Disease With Colovesical Fistula And Pericolonic Abscess

***DR. DONALD GRAHAM:** First of all, I think Dr. Olsen did an excellent job in his discussion of diverticular disease and its complications. Colovesical fistula is only one of the complications of diverticulitis for which we are called upon to do surgery, the others being obstruction, perforation with peritonitis, a non-resolving mass, bleeding, and intractability of symptoms.

As Dr. Olsen mentioned, the three-stage approach has been classically used to treat this problem. Often with a colovesical fistula, an abscess may be drained into the bladder, and if one is able to administer a good bowel prep, a one-stage procedure can be done safely with equal or less mortality and less morbidity. In some patients however, as was the case here, the inflammation can be so dense, that a colostomy is all that one can safely feel is indicated at that time. If possible, one should try to resect the disease. Whether a loop colostomy is performed depends upon the surgeon's confidence in the anastomosis.

****DR. A. J. HARTZELL:** I would like to congratulate Dr. Olsen on an excellent presentation. There are some problems in demonstrating a colovesical fistula. Sigmoidoscopy may be normal. I can remember a young man with Crohn's disease who had recurrent urinary tract infections over a two year period, but we could not demonstrate a colovesical fistula until the end of that time. Occasionally, the fistula may be easily demonstrated but this is more the exception than the rule. I like your comments concerning the visual dyes since demonstration of the fistula even with the more liquid contrast media used for barium enema or urograms is difficult. The bladder mucosa on cystoscopy often is markedly edematous and friable. We, of course, do biopsies to rule out carcinoma in the fistula but visualization of the fistula is often impossible. Pressure on the abdomen to demonstrate gas coming through the fistula is not usually helpful. We will often put dye into the bladder and place a sponge in the rectum to try to demonstrate the fistula.

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Peculiar things may happen. I remember one patient who gave a history of abdominal pain which only kept him out of work a few hours. He developed recurrent urinary tract infections and after some time I was able to demonstrate a fistulous tract which I injected obtaining an outline of the cecum and appendix. The patient had apparently had acute appendicitis and developed a fistula into the bladder and only been out of work for a few hours.

*****DR. JOHN BARKER:** I would like to mention that we are seeing diverticular disease in much younger patients. I would also like to ask Dr. Graham about the type of treatment a patient should have after he has recovered from an attack of acute diverticulitis.

DR. GRAHAM: The surgical indications for bleeding, perforation, obstruction, and fistula formation are more well-defined than those for intractability and diverticulitis. As in ulcer disease, intractability in diverticular disease has somewhat controversial indications for surgery. One reason for this is the difficulties in distinguishing attacks of mild diverticulitis from irritable bowel conditions. Generally, I think most surgeons would perform an elective sigmoid resection of the diverticula after two episodes of documented diverticulitis. In the case of severe diverticulitis in which a mass is present on physical examination, I think many people would perform an elective resection after this episode, as it seems these people go on to have more severe complications from their diverticular disease.

Certainly if there is any doubt at all that one is not dealing with diverticulitis, surgery should not be delayed.

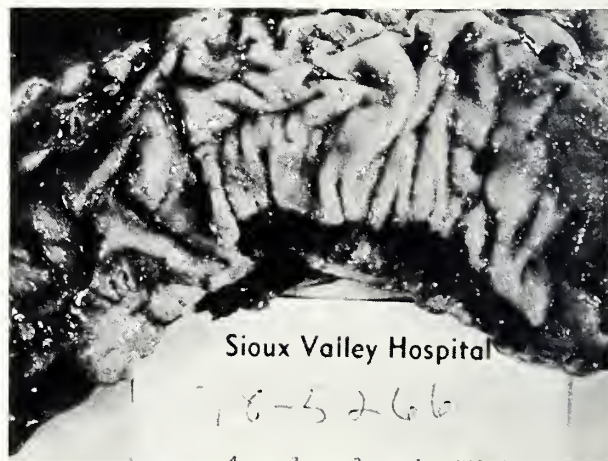


Figure 3

Note irregular thickening of colonic muscle and opening of diverticula in mucosa.

DR. BARLOW: This is a picture of the resected specimen. (Fig. 3) One can note the thickening of the muscle of the colon at the lower part of the pic-



Figure 4

Colonic mucosa penetrating muscular wall as incipient diverticulum. There is thickening and inflammation of serosa secondary to perforation of an adjacent diverticulum (not in picture).

ture and the diverticular openings on the mucosa. Also recognizable is the concertina effect which corresponds to the segmentation of the bowel in this region as described by Dr. Olsen. This photomicrograph (Fig. 4) shows portions of a diverticula penetrating through the wall of the colon. The serosa is thickened and inflamed secondary to diverticulitis of another diverticulum not shown in this picture.

FINAL ANATOMIC DIAGNOSIS

DIVERTICULAR DISEASE WITH COLOVESICAL FISTULA AND PERICOLONIC ABSCESS

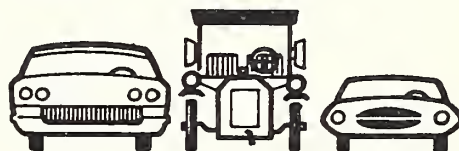
*DR. ENSBERG: I want to stress as it has been stressed by Dr. Graham and Dr. Olsen what you do with the patient who has a complication of diverticular disease depends very much on the findings at surgery. Also I question whether all cases of complicated diverticulitis should have a colon resection during a quiescent stage. Certainly a patient who has potential or actual fistula formation with the bladder or other organs should have a colon resection. However in other cases, I think the patient should be carefully informed and be brought into any decisions regarding resection. The clinical symptoms of the patient are extremely important.

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The Maker

Examining a Few Myths About Prescribing.

Increasing pressure is being put on the practicing physician to prescribe drugs generically. You are told that brand-name products are universally “expensive” and generic versions are relatively “cheap.” To make this case, the most extreme (rather than typical) price differentials are cited. Thus, consumers are led to believe that such differentials are commonplace. Even your knowledge and your motives as a physician are questioned.

Understandably, these views have created myths. We think it's time to examine them in the light of all the facts and ramifications.



MYTH: There are no differences in quality and performance between brand-name products and their generic counterparts. The corollary is that there are no differences among products made by high-technology, quality-conscious, research-based companies and those made by commodity-type suppliers.

FACT: The Food and Drug Administration does a good job in monitoring a generally excellent drug supply. Still, it has nowhere near the resources to guarantee the quality and bioavailability of all marketed products at any given time. Just a few months ago, for example, it noted that batches of tetracycline HCl capsules which met official monograph requirements were

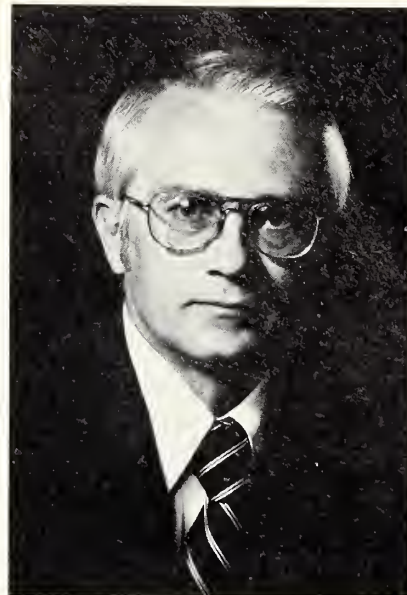
not bioequivalent to a reference product. As you know, there is substantial literature on this subject affecting many drugs, including such antibiotics as tetracycline and erythromycin. The record on drug recalls and court actions affirms strongly that there are differences among pharmaceutical companies and their products. Research-intensive companies have far better records than those that do no research and may practice minimum quality assurance.

MYTH: Industry favors only “expensive” brand names and denigrates all generics.

FACT: PMA companies make 90 to 95 percent of the drug supply, including, therefore, most of the generics. Drug nomenclature is not the important point; it's the competence of the manufacturer and the integrity of the product that count.

S **D**

President's Page



The nature and purpose of the practice of medicine has not changed from its beginnings. It remains, in general terms, the practice of the healing arts and sciences for the benefit of the people at large.

By the same token, the nature and purpose of the Medical Association has not changed. It was formed for the purpose of educating physicians and the populace regarding good medical practices. The thrust of this education process has, however, certainly broadened in recent years with much more emphasis being placed on social, economic and political activities as they pertain to medical affairs. These are legitimate and necessary areas of concern for your Medical Association as they affect the quality and availability of appropriate and adequate medical service to the public.

In early years, the AMA and state medical associations were primarily and appropriately concerned with education as it pertained to specific disease processes, but with the advent of the many specialty societies who have assumed a large share of that specific role, the AMA and the state societies have been able to devote much more time to the important socio-economic implications in the delivery of quality medical and, in a broader sense, health care to the people. It would be difficult, if not impossible, for this role to be assumed by the specialty societies because of their fragmented nature. It

therefore follows that both are needed, and both must be supported if we, as physicians, are to fulfill our obligations to the people in regard to medical affairs. This does not mean to say that the scientific aspects of the educational process should be abandoned by the AMA and the State Medical Association, but that a shift in emphasis is both necessary and appropriate. The umbrella nature of these organizations encompasses all of the varying viewpoints and thereby allows a consensus voice to speak for medicine at large, which is always more effective in accomplishing common goals and the public is sure to benefit. Your association needs you and wants your participation. I urge you all to continue your strong support.

It has been a rare privilege to serve as your president this past year. I thank you for allowing me to do so. Your staff continues to be first class and completely devoted to the activities of the State Medical Association and deserve your individual thanks the next time you see them.

Duane Reaney will be your new president and I know our association will flourish under his leadership and guidance. I wish him well.

In closing, I would again like to remind you that the physicians of this country have always been the people's advocate in medical affairs and would urge each of you to continue to be that advocate.

Fraternally yours,
Russell H. Harris, M.D., President
South Dakota State Medical Association

SD

Letters To The Editor

This letter is to inform you of the availability of the recently compiled **Directory of Rehabilitation Services for Brain Damaged Individuals in South Dakota and Outlying States**. This project was completed at the University of South Dakota Speech and Hearing Center in an effort to learn more about the various rehabilitation services available to South Dakota residents.

Included in the directory is background information of the project, procedures used to obtain the information, and a list of the different rehabilitation service providers including the types of services provided, facility capacities, and some of the more common rehabilitation techniques used in Speech Therapy.

We feel this directory would be beneficial to anyone involved in the rehabilitation of brain damaged individuals. Copies can be obtained from:

Dr. Sylvester Clifford
USD Speech and Hearing Center
University of South Dakota
Vermillion, South Dakota 57069

or

Jim Collings
Box 492
Broadus, Montana 59317

There will be a minimal charge to cover the costs of copying the material.

Sincerely,
Gail Wanthlin, M.A.
Jim Collings, M.A.
Speech Pathologists

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CONTRAINDICATIONS Hypersensitivity to oxycodone or acetaminophen.

WARNINGS **Drug Dependence** Oxycodone can produce drug dependence of the morphine type and, therefore, has the potential for being abused. Psychic dependence, physical dependence and tolerance may develop upon repeated administration of PERCOCET®-5, and it should be prescribed and administered with the same degree of caution appropriate to the use of other oral narcotic-containing medications. Like other narcotic-containing medications, PERCOCET®-5 is subject to the Federal Controlled Substances Act.

Usage in ambulatory patients Oxycodone may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. The patient using PERCOCET®-5 should be cautioned accordingly.

Interaction with other central nervous system depressants Patients receiving other narcotic analgesics, general anesthetics, phenothiazines, other tranquilizers, sedative-hypnotics or other CNS depressants (including alcohol) concomitantly with PERCOCET®-5 may exhibit an additive CNS depression. When such combined therapy is contemplated, the dose of one or both agents should be reduced.

Usage in pregnancy Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. Therefore, PERCOCET®-5 should not be used in pregnant women unless, in the judgment of the physician, the potential benefits outweigh the possible hazards.

Usage in children PERCOCET®-5 should not be administered to children.

PRECAUTIONS **Head injury and increased intracranial pressure** The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a pre-existing increase in intracranial pressure.

Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.

Acute abdominal conditions The administration of PERCOCET®-5 or other narcotics may obscure the diagnosis or clinical course in patients with acute abdominal conditions.

Special risk patients PERCOCET®-5 should be given with caution to certain patients such as the elderly or debilitated, and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, and prostatic hypertrophy or urethral stricture.

ADVERSE REACTIONS The most frequently observed adverse reactions include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients, and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include euphoria, dysphoria, constipation, skin rash and pruritus.

DOSAGE AND ADMINISTRATION Dosage should be adjusted according to the severity of the pain and the response of the patient. It may occasionally be necessary to exceed the usual dosage recommended below in cases of more severe pain or in those patients who have become tolerant to the analgesic effect of narcotics. PERCOCET®-5 is given orally. The usual adult dose is one tablet every 6 hours as needed for pain.

DRUG INTERACTIONS The CNS depressant effects of PERCOCET®-5 may be additive with that of other CNS depressants. See WARNINGS. 6085 BS

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EDO-644E 579

SD Chapter News



SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS
3001 South Holly Avenue
Sioux Falls, SD 57105



Spring Family Practice Club Activities

Under the direction of Chairman Howard J. Hoody of Sioux Falls, the SDAFP Family Practice Club will hold informal social events in Rapid City, Sioux Falls and Yankton.

Bill Tschetter and Gary Welsh of Rapid City will coordinate the west river event while Herb Saloum of Tyndall is in charge of the Yankton student social. Howard Hoody will handle the Sioux Falls arrangements.

These activities are being planned for all USDSM students in the area, with an emphasis on students in their third and fourth years. The fall and winter events are geared primarily for first and second year students.

An Admonition

In Boswell's *Life of Samuel Johnson* (1782) an admonition is given: "Cast away all anxiety and keep your mind easy. This (last) direction is the principal; with an unquiet mind, neither exercise, nor diet, nor physick can be of much use."

FP Resident Seeks Practice Opportunity

Ted Zdanowicz, M.D., a family practice resident in the Medical College of Ohio program in Toledo, is seeking a practice location beginning July 1, 1980.

His address is 1355 Glenview, Toledo, OH, 43614. He can be reached by phone at (419) 382-8074 (home) or at (419) 381-3568 (work).

A copy of his curriculum vitae is on file at the state academy office.

Family Physician Seeks SD Practice Site

John J. Tornow, M.D., a native of SD, is seeking a practice location in our state for 1980. He is now in the Air Force.

Dr. Tornow is FP residency trained and a Diplomate of the ABFP. His address is 3162 Altamonte Drive, Beale AFB, California 95903.

Board Candidate Nominated

The SDAFP nominating committee has placed Lawrence W. "Larry" Finney of Sioux Falls on the slate as a candidate for the Board of Directors. The election will be held at the Black Hills Summer Seminar August 10, 1979. Further nominations can be made from the floor at the annual business meeting.

ABFP Diplomates

The following list of SDAFP members were named Diplomates of the ABFP recently, having successfully completed the two day written exam held in testing centers throughout the United States in August of 1978:

Certified

Art J. Barrett, Rapid City
Christine F. Bucy, Beresford
Dennis L. Epp, Freeman
Harold J. Fletcher, Vermillion
Reginald S. Forshner, Hot Springs
Wm. Nicol Guddal, Watertown
Wm. O. Hanson, Huron
Louis H. Hogrefe, Gregory
Anthony J. Javurek, Deadwood
Peter E. Lakstigala, Sioux Falls
John A. Malm, Gregory
Ray Gene Nemer, Gregory
David A. Smith, Deadwood
John J. Stransky, Watertown
Lowell P. Swisher, Kadoka
T. J. Wrage, Jr., Watertown
David J. Yecha, Gettysburg
Arlan G. Zastrow, Huron

The following SDAFP members achieved recertification of their ABFP Diplomate status after sitting for a one day recertification examination.

Recertified

Bruce Lushbough, Brookings
Dennis J. Ortmeier, Sioux Falls
A. J. Tieszen, Pierre
Ray J. Zakahi, Pierre

Congratulations to these members. We now have 81 SDAFP Active members who are Diplomates, out of an active membership of 130.

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Decade
1969-1979*

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the
AAFP's Annual Scientific Assembly
October 8-11, 1979
With Special Family Events October 7-10
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1939 Cleo L. Vogeles, M.D.
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1944 M. J. Johnson, M.D.
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1948 C. H. Steele, M.D.
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1950 R. T. Orr, M.D.
1951 Phyllis Huffman, M.D.
1952 Dennis J. Walter, M.D.
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1956 C. W. Anderson, M.D.
1957 Lyle Munneke, M.D.
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1960 T. P. Gormley, M.D.
1961 Richard Porter, M.D.
1962 W. N. Gollither, M.D.
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1974 Steve Larson
1975
1976 Deborah R. Foley, M.D.
1977
1978 Robert Goodhope, M.D.

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...in the functional bowel/irritable bowel syndrome*

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In this double-blind study, twenty patients having G.I. series and exhibiting spasm were randomly selected to receive either 2 cc. of Bentyl or sodium chloride intramuscularly. Ten minutes after the injection another radiograph was taken . . .

. . . Bentyl produced definite relaxation in 8 of 10 patients. The sodium chloride produced relaxation in only 3 of 10. No side effects occurred in either group of patients.



Pylorospasm has almost totally blocked passage of barium meal.



Barium meal beginning to pass 10 minutes after intramuscular injection of 20 mg. Bentyl.

"The correlation of spasm relief and drug given was excellent."

*This drug has been classified "probably" effective in treating functional bowel/irritable bowel syndrome.

†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:

King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

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Capsules, Tablets, Syrup, Injection

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

INDICATIONS

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the following indications as "probably" effective:

For the treatment of functional bowel/irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

THESE FUNCTIONAL DISORDERS ARE OFTEN RELIEVED BY VARYING COMBINATIONS OF SEDATIVE, REASSURANCE, PHYSICIAN INTEREST, AMELIORATION OF ENVIRONMENTAL FACTORS.

For use in the treatment of infant colic (syrup).

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloroduodenal stenosis); paralytic ileus, intestinal atony of the elderly or debilitated patient; unstable cardiovascular status in acute hemorrhage; severe ulcerative colitis; toxic megacolon complicating ulcerative colitis; myasthenia gravis. **WARNINGS:** In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. **PRECAUTIONS:** Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy. Use with caution in patients with Autonomic neuropathy. Hepatic or renal disease. Ulcerative colitis. Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon. Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension. Hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.

Do not rely on the use of the drug in the presence of complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. **ADVERSE REACTIONS:** Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia; urinary hesitancy and retention; blurred vision and tachycardia; palpitations; mydriasis; cycloplegia; increased ocular tension; loss of taste; headache; nervousness; drowsiness; weakness; dizziness; insomnia; nausea; vomiting; impotence; suppression of lactation; constipation; bloated feeling; severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating. With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. **DOSEAGE AND ADMINISTRATION:** Dosage must be adjusted to individual patient's needs.

Usual Dosage: Bentyl 10 mg. capsule and syrup: Adults: 1 or 2 capsules or teaspoonfuls syrup three or four times daily. Children: 1 capsule or teaspoonful syrup three or four times daily. Infants: ½ teaspoonful syrup three or four times daily. (May be diluted with equal volume of water.) Bentyl 20 mg. Adults: 1 tablet three or four times daily. Bentyl Injection: Adults: 2 ml. (20 mg.) every four to six hours intramuscularly only. NOT FOR INTRAVENOUS USE. **MANAGEMENT OF OVERDOSE:** The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal. Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested. If indicated, parenteral cholinergic agents such as Urecholine® (bethanechol chloride USP) should be used.

Product Information as of October, 1978.

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D****Laboratory Aids****Sponsored by the South Dakota Society of Pathologists****Glycosylated Hemoglobin Levels
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The minor hemoglobin fractions, hemoglobin A_{1a}, hemoglobin A_{1b} and hemoglobin A_{1c} are referred to as a glycosylated hemoglobins, "fast" hemoglobins or glycohemoglobins.

The amino terminal valine residues of both beta chains of these hemoglobins are linked to hexoses. The higher the concentration of circulating glucose during the lifetime of the red blood cell (approximately 120 days) the higher the glycohemoglobin concentration. Originally, the glycohemoglobin Hb A_{1c} was thought to be more specific but the total glycohemoglobins including A_{1b} and hemoglobin A_{1a} show the same correlation as the hemoglobin A_{1c}.

METHOD OF QUANTITATION: The most commonly employed method is a cation exchange chromatographic technique. Thus the glycosylated hemoglobins can be quantitated quite rapidly and simply. The concept is of binding non-glycosylated hemoglobins more tightly to the column and allowing the fast fraction or glycosylated hemoglobins to be eluted first, measured, quantitated and then related to the remaining hemoglobins which are removed later. Then the glycosylated hemoglobin percentage is calculated.

Some care should be taken that the ambient "room" temperature be monitored with allowance for major shifts.

EXPECTED RANGE: The total glycosylated hemoglobin range in normal non-diabetic patients is 5.8-8.9%.

In diabetics requiring insulin, values greater than 10 and up to 22% have been recorded.

INTERPRETATION: The pertinent literature* states that glycohemoglobins can be used as an effective tool in monitoring the degree of control and in effect the average blood glucose concentration over the previous weeks to months in a diabetic.

This assumes that the goal of long term treatment is to maintain a blood glucose level in "normal ranges".

A distinct advantage of this procedure is that a non-fasting sample is valid.

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Barry T. Pitt-Hart, M.D.

Pathologist

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Intramural Hematoma Of The Duodenum

A Case Report And Literature Review

Bruce C. Lushbough, M.D.* D.A.B.F.P.

Saul Friefeld, M.D.** F.A.C.R.

Joseph Primrose, M.D. F.A.C.S.***

ABSTRACT

This is the case history of a 12 year old female who sustained a fall onto a fence post resulting in abdominal pain and vomiting and a diagnosis of intramural hematoma of the duodenum. Emphasis is on the susceptibility of the duodenum to injury because of its fixed position, and on the radiographic findings. A conservative approach is recommended by the authors, unless clinical signs demand surgical intervention.

CASE PRESENTATION

A 12 year-old female who was admitted to the Brookings Hospital was complaining of severe abdominal pain and vomiting which started after a fall from a fence. She had landed on the top of a fence post which struck her in the upper abdomen.

Physical examination in the Brookings Hospital emergency room showed normal vital signs, pulse of 100 and respirations of 24. The abdomen was soft with a questionable sensation of fullness just to the right of the midline of the epigastrium, which on initial examination seemed to be a superficial hematoma of the abdominal wall. Her white count was 12,000 with a normal amylase. She was subsequently placed in the hospital. Over the next six hours her abdominal pain increased in intensity with repeated

vomiting. Twelve hours post-admission her amylase was 786 and white count was now at 15,000 with unchanged physical findings.

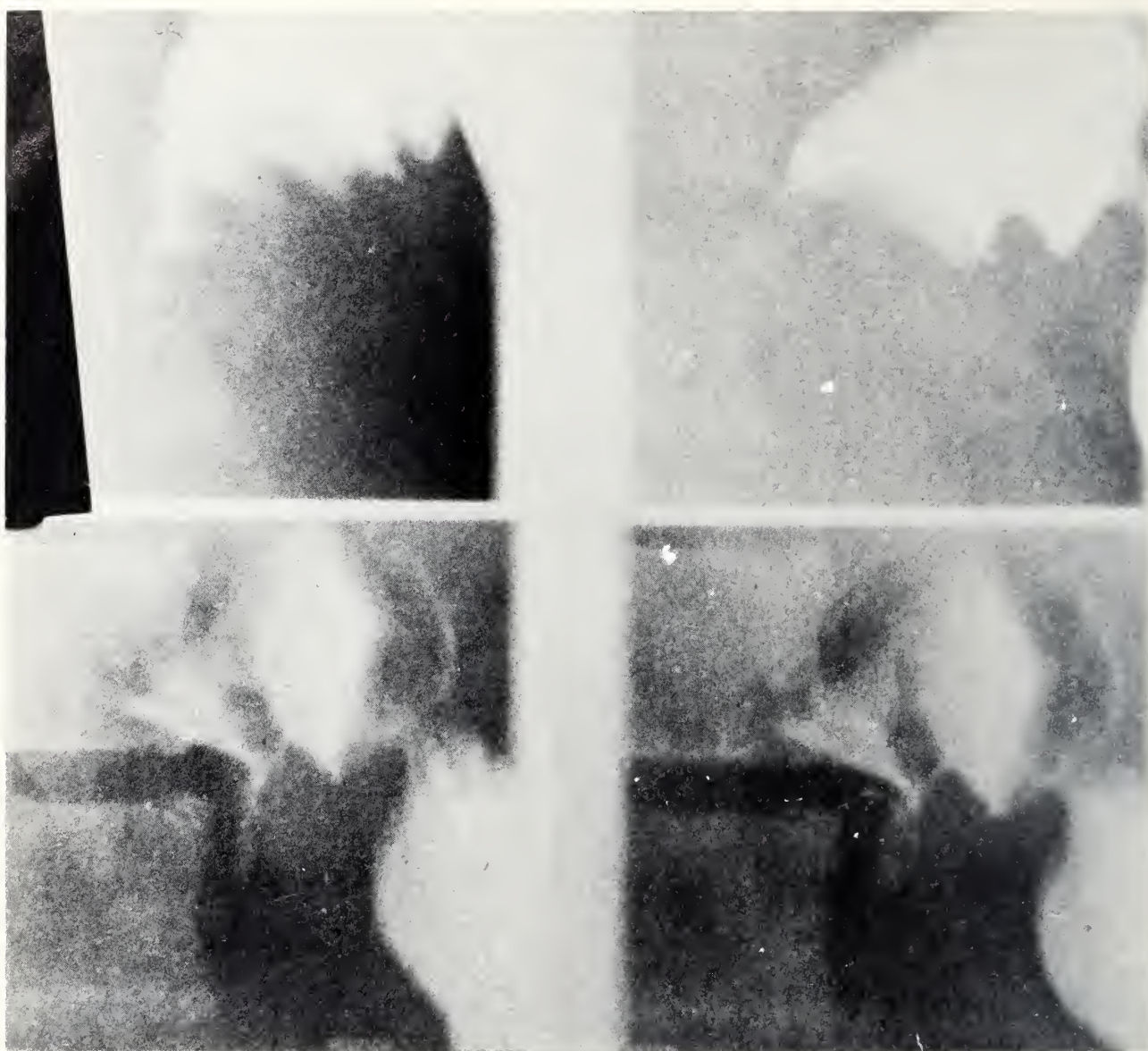
Subsequent flat and upright films of her abdomen and upper G.I. as seen by the accompanying slide, showed complete obstruction at the upper second portion of the duodenum with a spiral-like defect of the duodenum on upper G.I. It was noted at this time, there was no evidence of retroperitoneal leak of barium or evidence of peri-renal air. In the upper G.I. exam it is felt advisable to use barium instead of water soluble contrast medium, because of the dilution of the latter, and the much better delineation of the mucosal fold pattern with the barium.

She was maintained on I.V.'s and nasogastric suction for the next three days with gradual improvement in her vital signs and abdominal findings and symptoms. By the fifth day the patient was able to be started on gradual diet advancement and subsequent repeat upper G.I. studies showed gradual clearing of the intramural hematoma, with complete

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** Radiology, Brookings Hospital and Brookings Clinic, P.A., Brookings, SD.

***General Surgery, Brookings Hospital and Brookings Clinic, P.A., Brookings, SD.



resolution of the hematoma at two weeks post-injury.

DISCUSSION

As noted by Orr and Erlandson¹ in their review of literature in 1963, only 31 cases of intramural hematoma of the duodenum had been reported until that time. Intramural hematoma of the duodenum most often occurs as a result of blunt trauma and is frequently overlooked even at the time of exploratory laparotomy. The anatomical fixation of the duodenum makes it particularly vulnerable to injury, which at times may completely transect the duodenum or in lesser injury may give rise to a hematoma in the subserous or submucosa layers. As it enlarges, it results in compression of the mucosa and opposing walls of the duodenum, leading to complete obstruction of the duodenum and at times complete obstruction of the common bile duct.

The anatomic relationship of the duodenum with the pancreas results in injury frequently to both organs, especially in the alcoholic patients where the incidence of pancreatic disease and injury is higher.

The increased use of anticoagulants in the treatment of cardiac and vascular disease may possibly cause a rise in the incidence of hemorrhage into the duodenum as well as other parts of the body and they can also be initiated by such diseases as carcinoma and pancreatitis.

The majority of hematomas occur in children with an overwhelming majority of these having a history of trauma.

The frequent emesis and vomiting in the traumatized patient who has had no previous history of emesis and otherwise appears well and has few abnormal physical and laboratory findings should give rise to suspicion of this condition.

Diagnosis of intramural hematoma requires a high index of suspicion but without roentgenographic examination, an accurate diagnosis is often most difficult. Roentgenographic findings are always the most helpful and the classic coiled-spring appearance first described by Felson and Levine in 1954 is pathognomonic of the injury. As late as 1966 the surgical treatment of this disease consisted of laparotomy, evacuation of the duodenal hematoma and closure of the defect in the submucosa or seromuscular coat of the duodenum. In fact, as late as 1973, Shires and Ballinger^{2,3} recommended that the treatment of choice be laparotomy, complete assessment of the duodenal damage and a search for associated injuries with evacuation of the hematoma and duodenal decompression and even occasionally a gastroenterostomy.⁵

However, recently the more conservative approach has been taken, as described by Walt and Wilson,⁴ once other associated abdominal injuries have been ruled out. Maintenance of these patients on I.V. fluids and gastric decompression results in an excellent outcome. Close supervision of this conservative approach is required and a high index of suspicion maintained for other associated abdominal injuries and when these become clinically evident, an exploratory laparotomy should be immediately carried out.

SUMMARY

Intramural hematoma of the duodenum has infrequently been reported in the literature although in the past decade we have become aware of this injury much more frequently than in previous years because of a better understanding of blunt abdominal injuries. Minimal physical findings with repeated emesis and a history of upper abdominal trauma are most often associated with this entity. Subsequent confirmation by x-ray is mandatory. A high index of suspicion for other injuries of the upper abdomen and the duodenal area needs to be maintained.

In an otherwise uncomplicated duodenal injury when a coiled-spring appearance on upper G.I. is firm, a conservative approach results in excellent recovery, although exploratory laparotomy should be undertaken at the first sign of a deteriorating clinical course.

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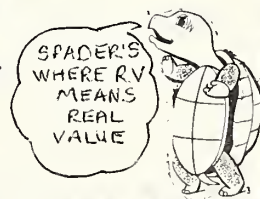


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Auxiliary Activities For 1978-79

South Dakota State Medical Association Auxiliary members have been involved in many areas of community health education, community service and legislation awareness. Throughout the state, auxiliaries participate in health related programs, meals-on-wheels, ecology awareness, medical career days at high schools, working with hospital auxiliaries, fund raising projects for AMA-ERF and child abuse and neglect awareness . . . besides the various service organizations, civic clubs and church groups where the physicians' spouses are prominent as leaders and workers.

There are two special projects that deserve recognition. They are in Aberdeen and Sioux Falls.

The Fortunate Four Program in Aberdeen (Verbal Auditory Screening for Pre-School Children) is financially sponsored by the District I Medical Auxiliary with the endorsement of South Dakota State Medical Association. At present time the Aberdeen Jayettes are coordinators and supervising administration of the survey. The district nurses have served as past coordinators. Many medical auxiliary members serve as volunteer workers.

In 1972 the Medical Auxiliary purchased a VASC audiometer with donated funds from the District I Medical Association and other local service organizations. The program is approved and supported by the schools and many civic organizations throughout the Aberdeen area. The survey depends specifically on voluntary contributions from individuals and organizations.

The first step of the survey is to obtain a census of all pre-school age children from local school officials. Organized volunteers receive instruction in screening procedures in a workshop conducted by an authorized demonstrator from the St. Paul office of the Minnesota Pre-School Medical Survey of Vision and Hearing. This program was developed and sponsored by the Minnesota State Medical Association.

Since 1972 thousands of Aberdeen area children have been screened for possible hearing and vision defects. In addition to identifying children in need of medical care and rehabilitation, sharpening the tools by which they learn, and furthering parent

education, communities which sponsor surveys will be making a solid contribution to the emotional health of these children.

The Child Protection Team, which consists of concerned agencies and individuals in Sioux Falls, invited the Seventh District Auxiliary to have a liaison on the Team. Seventh District Auxiliary has served as a resource for the Team and has been supportive of the Team's programs in combating the problem of child abuse and neglect in the community.

The Children's Inn, sponsored by the Team, opened its doors in November 1977. The Children's Inn is a crisis center which is professionally staffed 24 hours a day. It provides short-term housing for abused and neglected children and for mothers with children from the scene of domestic violence. The Inn is one of eight such centers in the country providing both shelter and counselling. Ten to fifteen cases a week of child abuse and neglect are reported to Social Services. The Inn has provided shelter for 218 children and 63 battered parents since its opening. The average stay for the children at the Inn is 5.4 days. As of September 1978 the Inn became autonomous. The Board of Directors of the Children's Inn consists of professionals, local business men and women and is receiving great support from the community. The first year the professionals were salaried from a Public Service Employment Grant. In January 1979 the Children's Inn tied into the United Way. Other revenues are available from the State Office of Children and Youth, partial contracting of services from the State and donations from community groups, including the Seventh District Auxiliary.

Auxiliaries, trained as extended family volunteers, participate as volunteers at the Inn, supervise the children's nursery during the Parents Anonymous meetings, which meets one morning a week and have acted as a supportive friend and confidante in potentially abusive family situations.

The Auxiliary members, with their increasing community activities and knowledge about health concerns, are working for better public relations and the improved image of medicine.

Mrs. Robert Van Demark
State Auxiliary President 1978-79

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Description: Each Anusol-HC Suppository contains hydrocortisone acetate, 10.0 mg; bismuth subgallate, 2.25%; bismuth resorcin compound, 1.75%; benzyl benzoate, 1.2%; Peruvian balsam, 1.8%; zinc oxide, 11.0%; also contains the following inactive ingredients: bismuth suboxide, calcium phosphate, and certified coloring in a hydrogenated vegetable oil base.

Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, bismuth suboxide, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Indications: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas and relief of local pain and discomfort following anorectal surgery.

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Contraindications: Anusol-HC[®] Suppositories and Anusol-HC[®] Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparation.

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Laboratory Medicine Update 1979, IDS Center Conf. Theatre, Minneapolis, MN, May 23-25. Fee: \$200. Category I credits. Contact: Office of CME, Box 293 Mayo Memorial Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Selected Topics in Internal Medicine, Boys Town Institute Aud., Omaha, NE, May 30-June 2. 20 hrs. AMA Category I and AAFP prescribed credits. Contact: Hattie DeLapp, CME, Creighton U., 2500 California St., Omaha, NE 68178.

Practical Dermatology in Primary Care, Mayo Memorial Aud., U. of Minn., Minneapolis, MN, May 31-June 2. Fee: \$150. Category I credits. Contact: Office of CME, Box 293 Mayo Memorial Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

June

Clinical Hypnosis, Earle Brown Con. Ed. Center, St. Paul, MN, June 1-2. Fee: \$150. Category I credits. Contact: Office of CME, Box 293 Mayo Memorial Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Contemporary Therapy of Cardiovascular Disease, Cragun's, Brainerd, MN, June 1-3. Fee: \$175—members of Am. Heart Assoc., \$225—non members. Category I credits. Contact: Office of CME, Box 293 Mayo Memorial Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Seminar for Family Physicians, U. of Vienna, Vienna, Austria, June 4-8. Contact: M. Arthur Kline, M.D., American Med. Soc. of Vienna, Lazarettgasse 13, Vienna IX, Austria.

Workshop on Prevention of Coronary Heart Disease, Spring Hill Center, Wayzata, MN, June 6-8. Fee: \$225. Category I credits. Contact: Office of CME, Box 293 Mayo Memorial Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

Incoordination, V.A. Hospital, Fort Meade, SD, June 7. 1 hr. AMA Category I credit. Contact: Office of Cont. Health Ed., USD, 800 E. 21st St., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Advances in the Treatment of Pain, V.A. Hosp., Hot Springs, SD, June 8. 1 hr. AMA Category I credit. Contact: Office of Cont. Health Ed., USD, 800 E. 21st St., Sioux Falls, SD 57101. Phone: (605) 339-7573.

South Dakota State Medical Association Annual Meeting, Howard Johnson Motor Lodge, Rapid City, SD, June 8-10. Contact: SDSMA, 608 West Ave., North, Sioux Falls, SD 57104.

Management of Cerebral Palsy—June 11-16, **Optional Practicum**—June 18-22, Children's Rehab. Center, U. of Minn., Minneapolis, MN. Fee: \$235—first week, \$150—opt. second week. Category I credits. Contact: Office of CME, Box 293 Mayo Memorial Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

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A Seminar on Advances in Pediatrics, Sylvan Lake Resort, Custer, SD, June 20-22. 15 hrs AMA Category I credit. Contact: Office of Cont. Health Ed., USD, 800 E. 21st St., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Mental Health Care Distribution-Delivery of Services, V.A. Hosp., Fort Meade, SD, June 21. 1 hr. AMA Category I credit. Contact: Office of Cont. Health Ed., USD, 800 E. 21st St., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Natural Family Planning Seminar, Marriott Inn, Bloomington, MN,

June 21-22. Fee: \$100. Category I credits. Contact: Office of CME, Box 293 Mayo Memorial Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

July

Joint Meeting of American Society of Clinical Pathologists and College of American Pathologists, Washington, DC, July 7-14. Contact: American Society of Clinical Pathologists, 2100 W. Harrison St., Chicago, IL 60612.

Topics in Emergency Medicine, Colby College, Waterville, ME, July 8-12. 24 hrs. AMA Category I credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby Coll., Waterville, ME 04901.

Surgical Techniques, Colby College, Waterville, ME, July 17-20. 15 hrs. AMA Category I credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby Coll., Waterville, ME 04901.

Coronary Disease, Exercise Testing, and Cardiac Rehabilitation, Playboy Resort, Lake Geneva, WI, July 27-29. Fee: \$202. 13 hrs. AMA Category I credits. Contact: International Med., Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

August

Ophthalmology, Colby College, Waterville, ME, Aug. 5-9. AMA and AAFP credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby College, Waterville, ME 04901.

Black Hills Summer Seminar, Holiday Inn of the Northern Hills, Spearfish, SD, Aug. 9-11. AMA and AAFP credits. Contact: Black Hills Summer Seminar, c/o SDSMA, 608 West Ave., N., Sioux Falls, SD 57104.

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, Four Seasons, Lake of the Ozarks, MO, Aug. 10-12. 13 hrs. AMA Category I credits. Fee: \$215. Contact: Internat'l. Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Obstetrics and Gynecology, Colby College, Waterville, ME, Aug. 12-13. (continued)

16. AMA and AAFP credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby College, Waterville, ME 04901.

Nuclear Medicine, Colby College, Waterville, ME, Aug. 12-17. AMA and AAFP credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby College, Waterville, ME 04901.

Medical Staff Law and Bylaws, The Abbey on Lake Geneva, Lake Geneva, WI, Aug. 13-15. Fee: \$350. Contact: Registrar, Aspen Systems Corp., 20010 Century Blvd., Germantown, MD 20767

Great Debates in Otolaryngology, Washington Plaza Hotel, Seattle, WA, Aug. 16-18. Fee: \$285. 23 hrs. AMA Category I credits. Contact: CME, U. of Wash. School of Medicine, E-303 HSB, SC-50, Seattle, WA 98195.

Coronary Disease, Exercise Testing and Cardiac Rehabilitation, Dunfey's Lodge, Cape Cod, MA, Aug. 17-19. Fee: \$202. 13 hrs. AMA Category I credits. Contact: Internat'l Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, Inn on the Park, Toronto, Canada, Aug. 17-19. Fee: \$215. 13 hrs. AMA Category I credits. Contact: Internat'l Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Forensic Medicine, Colby College, Waterville, ME, Aug. 19-23. AMA and AAFP credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby College, Waterville, ME 04901.

Pulmonary Disease, Colby College, Waterville, ME, Aug. 21-23. AMA and AAFP credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby College, Waterville, ME 04901.

Mackinac Island Conference, The Grand Hotel, Mackinac Island, MI, Aug. 24-27. AMA Category I credits. Contact: Office of CE, Towsley Center for CME, U. of Michigan Med. School, Ann Arbor, MI 48109.

24th Annual Conference, American Institute of Ultrasound in Medicine, Montreal, Canada, Aug. 27-31. Contact: Am. Inst. of Ultrasound in

Medicine, 6161 N. May, Suite 278, Oklahoma City, OK 73112.

September

American Cancer Society, National Conference—Breast Cancer 1979, Waldorf-Astoria Hotel, New York, NY, Sept. 6-8. 16 hrs. AMA Category I credits. Contact: Breast Cancer Conf., Am. Cancer Soc., 777 Third Ave., New York, NY 10017.

Seminar for Family Physicians, U. of Vienna, Vienna, Austria, Sept. 17-21. Contact: M. Arthur Kline, M.D., American Med. Soc. of Vienna, Lazarettgasse 13, Vienna IX, Austria.

Coronary Disease, Exercise Testing and Cardiac Rehabilitation, Sheraton on the Wharf, San Francisco, CA, Sept. 28-30. Fee: \$202. 13 hrs. AMA Category I credits. Contact: Internat'l Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, Sahara Hotel, Las Vegas, NV, Sept. 28-30. Fee: \$215. 13 hrs. AMA Category I credits. Contact: Internat'l Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

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**Coverage Of A Full Thickness Heel Wound By A
Muscle Transposition Flap**

**Clinicopathological Conference
Twenty-Six Year Old Postpartum Female With
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Table of Contents: page 3

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SCIENTIFIC ARTICLES

- 7 Coverage Of A Full Thickness Heel Wound
By A Muscle Transposition Flap
William Lineaweaver, M.D.
Joyce Schultz, M.D.
- 23 Clinicopathological Conference
Twenty-Six Year Old Postpartum Female
With Thyrotoxicosis
Thomas Grau, M.D.
Paul K. Aspaas, Jr., M.D.
Fred Lovrien, M.D.
John F. Barlow, M.D.

FEATURES

- 10 President's Page
- 15 Laboratory Aids
South Dakota Pathology Page
- 17 South Dakota AFP Chapter News
- 21 This Is Your Medical Association
- 34 Letters To The Editor
- 37 Council Meeting Highlights
- 40 Future Meetings

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Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence:* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy:* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children:* Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdose. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular:* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System:* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal:* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic:* Urticaria, rash, ecchymosis, erythema. *Endocrine:* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System:* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous:* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in the evening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in the morning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdose include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdose.

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References: 1. Citations available on request — Medical Research Department, MERRELL RESEARCH CENTER, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon, R.H., and Leyland, H.M.: A Comprehensive Review of Diethylpropion Hydrochloride. International Symposium on Central Mechanisms of Anorectic Drugs, Florence, Italy, Jan. 20-21, 1977.

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Coverage Of A Full Thickness Heel Wound By A Muscle Transposition Flap

William Lineaweaver, M.D.*

Joyce Schultz, M.D.*

ABSTRACT

A defect resulting from a full thickness loss of heel pad tissue was reconstructed with a flexor digitorum brevis transposition flap. Principles of muscle flap use and details of the flexor pollicis brevis flap are discussed.

A full thickness loss of the tissue overlying the calcaneus present a problem of coverage and function. Whatever solution may be proposed must restore the integument in such a way that functional padding is provided.

Simple skin grafting is obviously inadequate. Techniques of cross leg flap coverage require long periods of tedious immobilization, and their very concept is compromised by having to introduce new tissue into an area with a poor blood supply. As discussed by Bostwick,¹ these flaps have not proven to be reliable long term solutions, probably because of their failure to thrive in such a relatively avascular site.

Recently, a patient presented to the Rosebud Indian Health Service Hospital with a full-thickness heel wound and we were prompted to explore reconstructive options. We elected to use a muscle transposition flap for coverage.

CASE REPORT

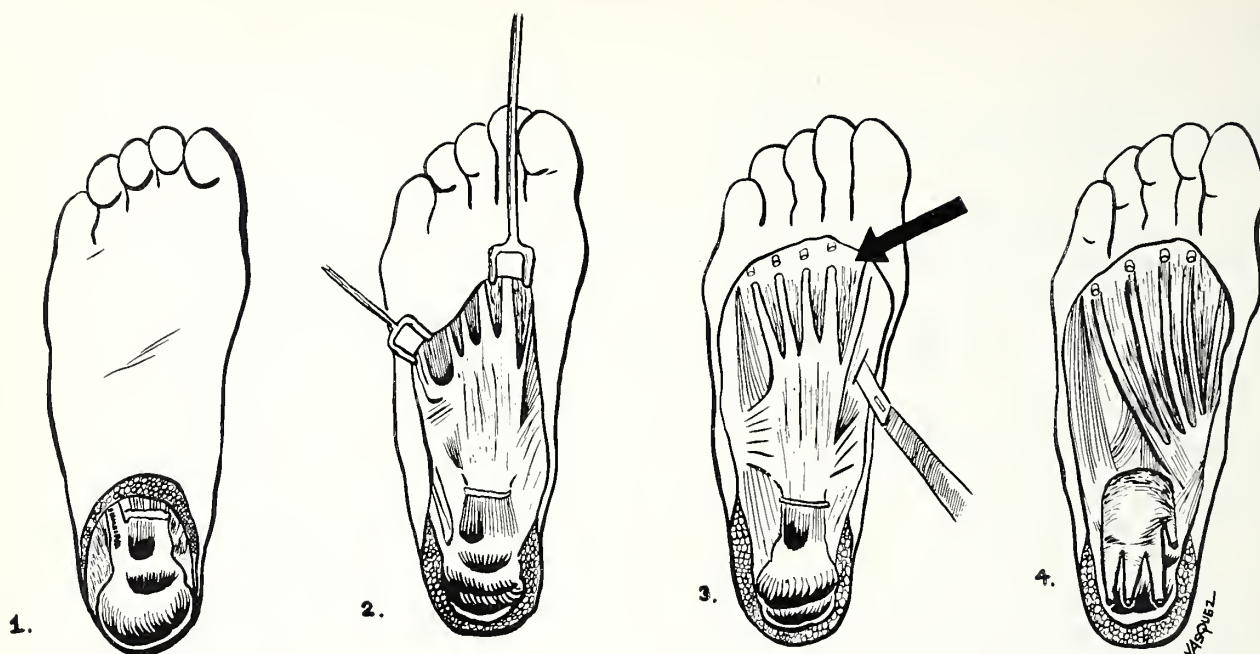
John E. was a 28 year old T-10 paraplegic who was admitted to the medical service on January 12, 1978, after a prolonged period of self neglect. At

that time, he was found to have a temperature of 103 F., was disoriented, and was further found to have pressure sores of his sacrum and both heels. His most immediate problem was that of a obstructed indwelling catheter, and, with replacement, he became afebrile. He was started on whirlpool and dressing treatment for his pressure sores. The sacral and right heel wounds were superficial, and cleared up promptly. The left heel wound showed little progress.

On January 20, he was transferred to the surgery service. The left heel was debrided and necrotic tissue was removed down to the plantar surface of the calcaneus. The wound finally consisted of a 3 cm defect of heel surface with a 2 cm base of exposed bone. After two weeks of topical care and nutritional support, the heel defect was closed with a flexor digitorum brevis muscle flap. Skin grafts were used to cover the surface of the flap and the right heel.

Postoperatively, the patient's course was uncomplicated except for superficial infections of the skin graft margins which responded to topical care. He remained in the hospital one month postoperatively, largely for social reasons, and the flap and overlying graft were well healed on discharge. He was seen in clinic two months postoperatively and the repair had remained intact.

*Indian Health Service Hospital, Rosebud, SD.



1. Heel wound with exposed calcaneus.
2. Exposure of aponeurosis.
3. Elevation of muscle with aponeurotic strip; arrow indicates divided tendons.
4. Insertion and closure.

TECHNIQUE

An incision was made from the medial margin of the heel pad wound along the medial aspect of the plantar surface to the base of the great toe. The incision was carried to the plantar aponeurosis. The flexor digitorum brevis was identified beneath the aponeurosis. Incisions were then made in the aponeurosis in such a way that the belly of the flexor digitorum brevis muscle could be bluntly freed while a strip of aponeurosis remained attached to the belly surface. The muscle's tendonous insertions were divided so as to leave about one centimeter of each tendon attached to the distal end of the muscle. The muscle was then folded back upon itself and laid into the wound so as to cover the calcaneus. The attached strip of aponeurosis was sutured to the juxtaposed proximal aponeurosis relieving the distal portion of the flap of any tension. The tendon stumps were sutured to the wound margin so that the muscle securely filled the defect. The plantar surface incision was closed and a skin graft was applied to the exposed muscle.

DISCUSSION

The problems posed by full thickness tissue losses

of the lower extremities were imaginatively addressed by Ger in the mid 1960's when he began to describe a series of muscle transposition techniques culminating in a detailed review published in 1972.²

These techniques offer the advantage of providing tissue bulk complete with blood supply to an open wound in a single procedure. The principles of designing the flaps are straight forward.^{3,4} Initially, the muscle belly chosen must be ample enough to reach and fill the defect. The blood supply to the muscle should be near the area which will remain attached to its anatomic position and hence would not be disrupted by the transposition. Finally, if the limb is still functional, the muscle chosen for transposing should be one whose loss will not result in a significant disability. Muscle transposition flaps have been described for various soft tissue defects of upper and lower extremities, head, chest, sacrum and perineum.⁴

The flexor digitorum brevis flap meets all requirements for heel wound coverage and has been used successfully in ambulatory and non-ambulatory patients.^{1,4}

The muscle itself originates at the medial aspect

of the calcaneal tuberosity and receives a branch of the lateral plantar artery near its origin. This blood supply is reported as being constant and adequate to preserve perfusion. Lateral phalangeal flexion is preserved by the flexor digitorum longus after transfer of the flexor brevis.^{3,5} The length and bulk of the muscle are sufficient to provide the tissue mass needed for heel pad reconstruction.

BIBLIOGRAPHY

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ASPEN MUSHROOM CONFERENCE

Identification of edible, poisonous and hallucinogenic mushrooms. Treatment of mushroom poisoning. Microscopy. Novice and advanced courses. AMA Category 1. July 29-August 3, 1979. Wildwood Inn, Snowmass-at-Aspen, CO.

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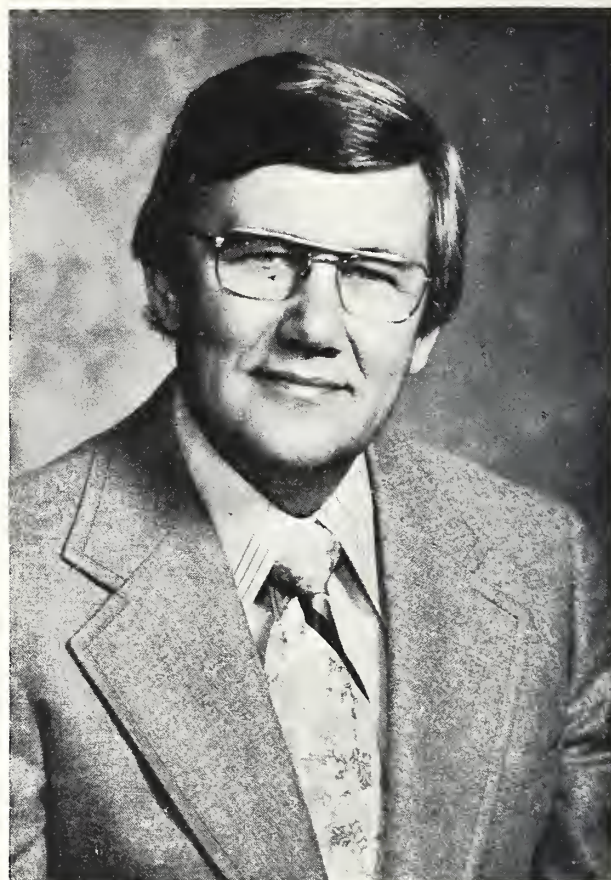


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S D

President's Page



The position of Presidency of the South Dakota Medical Association, seems, in many respects, to be awesome. I am looking forward to this challenge and pledge to the membership my best efforts to serve you well.

Any President in recent years, has had a lot going for him. Our executive secretary and his fine staff have given great support to the office of the President. It is obvious that the membership in general

has been generous in supporting the many endeavors of the association.

I will be asking many of you, individually, to give of your talents to aid the association. Naturally, the support of the general membership is always necessary. With all of us working together, I am sure your State Medical Association will again serve you well in the ensuing year. I hope all members will feel free to convey their problems and concerns to me, the council, or our staff.

Sincerely,
D. B. Reaney, M.D., President
South Dakota State Medical Association

SD Laboratory Aids

Sponsored by the South Dakota Society of Pathologists

Monoclonal Gammopathy

In general a monoclonal gammopathy is detected by performance of electrophoresis of a patient's serum proteins usually because the physician has discovered that the patient has an elevated total serum protein concentration, or the serum albumin is reduced and it appears that the globulin fraction of the proteins is increased. Visual inspection of the electrophoretic pattern in such patients shows a single spike in the serum globulin usually occurring in the beta or gamma, rarely in the alpha region. The globulin peak in a monoclonal gammopathy is characterized by a high, narrow spike which rivals the albumin spike on the electrophoretogram.

The nature of this spike should be clarified by the use of immunoelectrophoresis which separates the immunoglobulins into five fractions, IgG, IgM, IgA, IgD and IgE; of the total immunoglobulins, IG represents 80%. These immunoglobulin fractions are produced by plasma cells or mature B cells. The immunoglobulin fractions can be quantitated by radial immunodiffusion techniques. These studies should be done on serum and urine.

In patients with multiple myeloma, immunoelectrophoresis reveals an increase in IgG, IgA or IgD immunoglobulins which are believed to be the result of one clone of plasma cells producing an excessive amount of a homogeneous immunoglobulin. In macroglobulinemia of Waldenstrom, IgM immunoglobulins are produced in excess. In both multiple myeloma and macroglobulinemia, Bence-Jones protein may be present in the urine, and is detected as a narrow spike in the electrophoretogram of urine. Bence-Jones protein has a low molecular weight and is composed of light chains present in excessive amounts. Normally each immunoglobulin molecule is composed of two light chains and two heavy chains of polypeptides.

Most patients with multiple myeloma and macroglobulinemia exhibit a normochromic, normocytic anemia; platelet counts may be normal or depressed; and the leukocyte count may be normal or depressed. Occasional plasma cells may be seen in the peripheral blood of patients with multiple myeloma. Bone marrow examination in multiple myeloma reveals proliferations of plasma cells which may appear to

be mature, reactive or neoplastic, or may resemble lymphocytes. These cells may vary from 20% to 90% of the nucleated cells in the bone marrow in well developed cases. In macroglobulinemia (Waldenstrom type), the marrow is hypercellular, revealing primarily proliferations of lymphocytes among which are mixed various types of plasma cells. X-ray examinations of the bones and bone scans may be needed to ascertain the presence of lesions which occur in multiple myeloma, but are rare in macroglobulinemia.

Patients who have heavy chain disease exhibit malignant proliferations of cells in the lymphoreticular system and present a lymphoma-like clinical picture. Here too the serum protein electrophoretogram exhibits a monoclonal spike as a result of the accumulation of fragments of IgG.

About one-third to one-half of all patients who have a monoclonal gammopathy do not have a malignant disease or the condition is associated with non-B cell disorders, and they have a life span considerably longer than those patients with multiple myeloma, macroglobulinemia or heavy chain disease. In patients who have progressive monoclonal gammopathies, the hemoglobin concentration usually is decreased and the erythrocytic sedimentation rate is increased sometime during the course of their disease whereas this is not true in those who have a benign type of monoclonal gammopathy. Furthermore in benign disease the bone marrow aspirate usually reveals less than 10% plasma cells. In benign monoclonal gammopathies the unaffected immunoglobulins are generally present in normal concentrations whereas in the malignant forms, the unaffected immunoglobulin may be depressed. Almost always the presence of Bence-Jones protein in the urine indicates a malignant monoclonal gammopathy. Furthermore, in patients with a malignant monoclonal gammopathy repeated measurements of the M protein whether due to multiple myeloma, macroglobulinemia or heavy chain disease reveals an unabated rise as the condition progresses. Monoclonal spikes may be seen in healthy, asymptomatic patients and occasionally in patients who have diabetes mellitus, cirrhosis, collagen disease or epithelial malignancies such as basal cell carcinoma and squamous cell carcinoma.

John T. Tidd, M.D.
Pathologist

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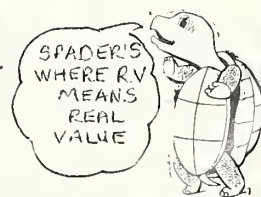


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WEDNESDAY, AUGUST 8, 1979

6:00 p.m. Board Meeting, SDAFP

THURSDAY, AUGUST 9, 1979

Morning Session

B.O. Lindbloom, M.D., Moderator

8:00- 9:00 Registration
9:00- 9:10 Welcome—B.O. Lindbloom, M.D.
9:15- 9:55 George C. Flora, M.D.
Seizures
10:00-10:15 Coffee
10:20-11:00 Larry T. Patterson, Pharm.D.
Coma, Drug Induced or Not?
11:05-11:45 George C. Flora, M.D.
Coma
12:00- 1:15 Hosted Luncheon—SDAFP
Memorial Lecture, *Your Body—Use It or Lose It*
James Ryan, M.D.

Afternoon Session

Wm. R. Tschetter, M.D., Moderator

1:30- 2:10 Larry T. Patterson, Pharm.D.
Drug Incompatibilities and Interactions
2:15- 2:55 George C. Flora, M.D.
Treatable Dementia
3:00- 3:15 Coffee and Iced Tea
3:20- 4:00 R. R. Lawrence, M.D.
Office Techniques in Minor Surgery
4:05- 4:55 Panel Discussion
5:00 Meeting—Legislative Committee, South Dakota Chapter, AAFP
Meeting—Education Committee, South Dakota Chapter, AAFP

FRIDAY, AUGUST 10, 1979

Morning Session

Earl Kemp, M.D., Moderator

8:00- 9:00 Registration
9:00- 9:40 George C. Flora, M.D.
Diagnosis and Treatment of Demyelinating Diseases
9:45-10:25 Larry T. Patterson, Pharm.D.
Common Poisonings Seen in the Emergency Room

10:30-10:45 Coffee
10:50-11:25 R. R. Lawrence, M.D.
Update: Therapy for Rattle Snake Bites
11:30-12:00 Panel Discussion
12:15- 1:15 Hosted Luncheon with a Speaker (select one)
George C. Flora, M.D.
Larry T. Patterson, Pharm.D.
R. R. Lawrence, M.D.
Helen Jane Hare, M.D.
Robert A. Green, M.D.

Afternoon Session

Michael Brown, M.D., Moderator

1:30- 2:05 Robert A. Green, M.D.
Chemotherapy of Leukemias and Lymphomas
2:05- 2:40 Helen Jane Hare, M.D.
Update on Acne, Seborrheic Dermatitis and Psoriasis
2:40- 3:15 R. R. Lawrence, M.D.
Diagnosis and Treatment of Heat and Cold Injuries
3:15- 3:30 Coffee and Iced Tea
3:30 Annual Business Meeting, South Dakota Chapter, AAFP
6:30 Cocktail Party and Dinner in the Hills

SATURDAY, AUGUST 11, 1979

Morning Session

James Ryan, M.D., Moderator

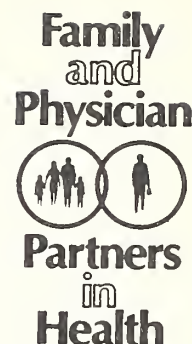
8:00- 9:00 Registration
9:00- 9:30 Robert A. Green, M.D.
Chemotherapy of Breast Cancer
9:35-10:05 Helen Jane Hare, M.D.
Diagnosis and Treatment of Malignant and Premalignant Skin Diseases
10:05-10:15 Coffee
10:15-10:45 Robert A. Green, M.D.
Chemotherapy of G.I. and Pulmonary Neoplasms
10:50-11:20 Helen Jane Hare, M.D.
Diagnosis and Treatment of Common Skin Infections
11:25-12:00 Panel Discussion

MAKE PLANS TO ATTEND NOW. WRITE: BLACK HILLS SUMMER SEMINAR
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SD Chapter News



SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS
3001 South Holly Avenue
Sioux Falls, SD 57105



Proposed Bylaw Changes/Additions

(To be voted upon at Annual Business Meeting
August 10, 1979)

Submitted by Board of Directors

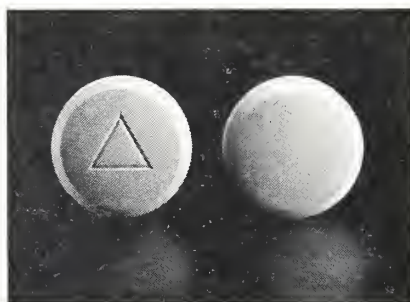
1. Bylaws, Chapter IV, Section 2. Deletes the last sentence reading: Remuneration for two delegates or alternate delegates attending the Congress of Delegates of the American Academy of Family Physicians shall include transportation and motel accommodations for said approved delegates. This is updated and covered in Chapter IX, Section 4.
2. Bylaws, Chapter V, Section 1. Deletes the last sentence reading: The president shall be reimbursed by the organization for transportation expenses to and from said meeting (ed. AAFP National Convention), and for the cost of hotel accommodations while attending said meeting. This is updated and covered in Chapter IX, Section 4.
3. Bylaws, Chapter VI, Section 2. This currently reads: The Board of Directors shall meet once annually at the annual meeting of the South Dakota Medical Association and such other time and places as the president may determine. Proposed changes would have Section 2 read as follows: The Board of Directors shall meet annually at the time of the annual business meeting of the South Dakota Academy of Family Physicians and shall meet one additional time during the business year, the time and location to be determined by the president and board of directors.
4. Bylaws, Chapter IX, Section 4. This currently reads: This organization's delegates to the Congress of Delegates of the American Academy of Family Physicians shall be reimbursed by this organization for transportation expenses to and from said meeting and for the cost of hotel accommodations while attending said meeting. All other expenses shall be borne by the individual delegates. Proposed changes would have Section 4 read as follows: This organization's president, during his presidential year, and delegates or alternate delegates (maximum of two) to the Congress of Delegates of the American Academy of Family Physicians shall be reimbursed by this organization for attendance at the Congress of Delegates and National Convention, AAFP, according to the following criteria: "equivalent" coach airfare plus eighty-five dollars (\$85) per diem, based upon one day for travel plus the official days set for the meeting. All other expenses shall be borne by the individual.
5. Bylaws, Chapter IX, Section 6. The secretary-treasurer, SDAFP, shall be reimbursed by the SD Chapter, SDAFP, for transportation, room, and board expenses incurred in attending the annual State Officers Conference and the annual National Convention of the AAFP. Proposed changes would have Section 6 read as follows: The secretary-treasurer, SDAFP, shall be reimbursed by the SD Chapter, SDAFP, for total meeting support, including registration, transportation, room, board, and incidental expenses incurred in attending the annual AAFP State Officers Conference and National Convention and all SDAFP sponsored scientific and business meetings, such as the Black Hills Seminars.
6. Bylaws, Chapter IX, Section 8. This currently reads: Resident representative to National Conference on Resident and Student Affairs, AAFP. The SDAFP will pay up to \$250 for attendance by an FP Resident in a South Dakota program to attend the annual meeting of resident representatives from State Chapters, sponsored by AAFP's Committee on Resident and Student Affairs. The Board of Directors, SDAFP, will select the resident to attend this annual meeting, acting upon the recommendation of the Director(s) of the Family Practice Residency Program(s). The resident may be selected more than one year in succession to attend this meeting. Proposed changes would have Section 8 read as follows: Resident representative to National Conference on Resident and Student Affairs, AAFP. The SDAFP will pay up to \$250 each for attendance by one or two FP Resident(s) from a South Dakota program to attend the annual meeting of resident representatives from State Chapters, sponsored by AAFP's Committee on Resident and Student Affairs. The Board of Directors, SDAFP, will select the resident(s) to attend this annual meeting, acting upon the recommendation of the Director(s) of the Family Practice Residency Program(s) in South Dakota. Each resident(s) may be selected more than one year in succession to attend this meeting.
7. Bylaws, Chapter IX, Section 11. Proposed addition to the Bylaws. Section 11 (new) would read as follows: One or two "designated" Family Practice Resident(s) from South Dakota program(s) will be allowed to sit with the Board of Directors, SDAFP. Resident(s) so designated are to be selected by their family practice residency program(s) and serve at their pleasure. No financial support or vote will be allowed for this activity.

The Maker

Examining a Few Myths About Prescribing.

Increasing pressure is being put on the practicing physician to prescribe drugs generically. You are told that brand-name products are universally “expensive” and generic versions are relatively “cheap.” To make this case, the most extreme (rather than typical) price differentials are cited. Thus, consumers are led to believe that such differentials are commonplace. Even your knowledge and your motives as a physician are questioned.

Understandably, these views have created myths. We think it's time to examine them in the light of all the facts and ramifications.



MYTH: There are no differences in quality and performance between brand-name products and their generic counterparts. The corollary is that there are no differences among products made by high-technology, quality-conscious, research-based companies and those made by commodity-type suppliers.

FACT: The Food and Drug Administration does a good job in monitoring a generally excellent drug supply. Still, it has nowhere near the resources to guarantee the quality and bioavailability of all marketed products at any given time. Just a few months ago, for example, it noted that batches of tetracycline HCl capsules which met official monograph requirements were

not bioequivalent to a reference product. As you know, there is substantial literature on this subject affecting many drugs, including such antibiotics as tetracycline and erythromycin. The record on drug recalls and court actions affirms strongly that there are differences among pharmaceutical companies and their products. Research-intensive companies have far better records than those that do no research and may practice minimum quality assurance.

MYTH: Industry favors only “expensive” brand names and denigrates all generics.

FACT: PMA companies make 90 to 95 percent of the drug supply, including, therefore, most of the generics. Drug nomenclature is not the important point; it's the competence of the manufacturer and the integrity of the product that count.

SD

This Is Your Medical Association

Bernard C. Gerber, M.D., Aberdeen, has been appointed a consulting editor for the surgical journal, **SURGERY, GYNECOLOGY AND OBSTETRICS**.

* * * *

Charley Gutch, M.D., Sioux Falls, presented a seminar on Renal Disease Dialysis: Long Term Care, for registered nurses at Augustana College.

* * * *

Dr. and Mrs. Edward Huppler, Watertown, won trophies at the Lake City Duplicate Bridge Club championship game. **David Piro, M.D.**, Watertown, placed sixth.

* * * *

Fred Lovrien, M.D., Sioux Falls endocrinologist, spoke on Thyroid Function Testing at a recent meeting of the Watertown District Medical Society.

* * * *

E. A. Johnson, M.D., Milbank, was presented the Distinguished Service Award of the South Dakota High School Activities Association in the category of Community Support.

* * * *

The Sioux Falls District Auxiliary presented a panel discussion at the Child Abuse Workshop held in Watertown. Participants included **Mrs. Jean Orr**, **Mrs. Bertie VanDemark**, **Mrs. Jan Cutshall** from Sioux Falls, and **Mrs. Bonnie Piro**, Watertown.

South Dakota physicians who have been named diplomates of the American Board of Family Practice include **Arthur Barrett, M.D.**, Rapid City; **Larry Mattson, M.D.**, Redfield; **W. Nick Guddal, M.D.**, John Stransky, M.D. and **T. J. Wrage, Jr., M.D.**, Watertown.

* * * *

Stephen Haas, M.D. presented a class on juvenile diabetes for diabetic children and their families at Rapid City Regional Hospital.

* * * *

Registered nurses attending a seminar at Augustana College heard **John Argabrite, M.D.**, Watertown, discuss Allergies.

Mrs. Bertie Van Demark, 1978-79 Auxiliary president, was the recipient of the civic and humanitarian affairs award presented at the sixth annual YMCA Leader Luncheon in Sioux Falls.

* * * *

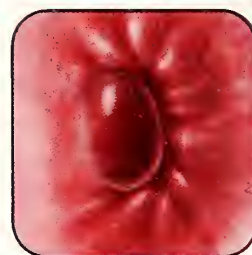
Speakers at recent Aberdeen District Medical Society meetings included **Robert Talley, M.D.**, Sioux Falls, who spoke on Pulmonary Embolism and **Ismael Unite, M.D.**, Aberdeen, whose presentation was on Selected Aspects of Radionuclide Scanning.

* * * *

Paul C. Reagan, M.D., 64, Sioux Falls, died April 21, in Des Moines, Iowa. Dr. Reagan was a graduate of the University of Iowa School of Medicine, he interned in Indianapolis, Indiana, and served with the Army Air Force in the United States and Korea. Following World War II he joined his father in the practice of medicine in Sioux Falls. Dr. Reagan is a past president of the Seventh District Medical Society, a charter member of the South Dakota Chapter, American Academy of Family Physicians, a member of the South Dakota State Medical Association and was active in civic and music activities in the community. He is survived by his wife, four daughters, one son, six grandchildren, two brothers and one sister.

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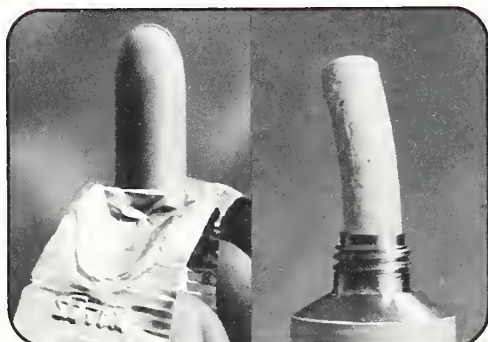
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Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, bismuth subiodide, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Indications: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain and discomfort in: external and internal hemorrhoids, proctitis, popliteitis, cryptitis, anal fissures, incomplete fistulas and relief of local pain and discomfort following anorectal surgery.

Anusol-HC Cream is also indicated for pruritus ani. Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol[®] Suppositories or Ointment.

Contraindications: Anusol-HC[®] Suppositories and Anusol-HC[®] Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparation.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts, or for prolonged periods of time.

Precautions: Symptomatic relief should not delay definitive diagnoses or treatment. If inflammation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Core should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Anusol-HC is not for ophthalmic use.

Dosage and Administration: Anusol-HC Suppositories—Adults: Remove foil wrapper and insert suppository into the anus. One suppository in the morning

and one at bedtime, for 3 to 6 days or until inflammation subsides. Then maintain patient comfort with regular Anusol Suppositories.

Anusol-HC Cream—Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain patient comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories—boxes of 12 (N 0047-0089-12) and 24 (N 0047-0089-24); in silver foil strips with Anusol-HC W/C printed in black.

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Twenty-Six Year Old Postpartum Female With Thyrotoxicosis

Thomas Grau, M.D.*

Paul K. Aspaas, Jr., M.D.**

Fred Lovrien, M.D.***

Discussers

John F. Barlow, M.D.****

Editor

Case No. 583125

This 26-year-old Caucasian female was referred to a specialist in internal medicine in the postpartum period because of fatigue and headaches.

Three months prior to admission, her obstetrician had noted that the thyroid had become diffusely enlarged but was non-tender. A T4 and T3 resin uptake were within normal limits. The patient noted palpitations and that her skin had become slightly more oily. She had lost a few pounds in weight but had been dieting. There was no change in her personality, emotions or bowel habits. She was taking no medications.

PHYSICAL EXAMINATION: Well developed young woman, temperature 98.6°F, pulse 88/minute and regular, respirations 16/minute and regular. Blood pressure 122 systolic and 66 diastolic in the left arm sitting. The skin was oily and somewhat smooth in texture. There was bilateral lid lag. The thyroid was two and a half times enlarged, diffuse, smooth, firm and non-tender. The chest was clear to auscultation and percussion. The cardiac size was normal and there was a soft flow murmur audible at the base. No scratch was audible. There was a fine tremor of the extremities. The neurologic examination was within normal limits with brisk deep tendon reflexes. Initial thyroid tests were as follows: thyrotrophin 3 IU/ml (normal less than 20 IU/ml), effective thyroxine ratio 1.24 (normal 0.90-1.17), T4 by radioimmunoassay 18 micrograms/dl (normal 5-4 mgs/dl); T3 by radioimmunoassay 420 mgs/dl (normal 60-190 mgs/dl). The patient was placed on propranolol which seemed to control her symptoms quite nicely. A

thyroid uptake and scan revealed only a 1% uptake in 24 hours and there was poor visualization of the thyroid outline on the scan. The patient was continued on propranolol and was gradually tapered off that medication. In the ensuing months, she gradually developed fatigue, lethargy, and started to gain weight. Her T4 was 1.3 mgs %; T3 resin uptake 25% (normal 22-35%) and thyrotrophin 62 IU/ml. The patient was then started on L-thyroxine which brought her T4 to 11.4 mgs/ul on a dose of 0.2 mgs of L-thyroxine each day.

DR. GRAU: Essentially, the picture presented is that of a young female in the postpartum period who, initially, clinically and by laboratory evaluation demonstrates hyperthyroidism and subsequently over a few months becomes hypothyroid.

There are several interesting aspects of this case, so I would like to spend the majority of the time discussing these. However, because of its importance in understanding thyroid dysfunction, I would first like to review briefly some very basic thyroid physiology and its relationship to thyroid function testing, paying particular attention to those tests mentioned in today's case.

The normal function of the thyroid gland is secretion of triiodothyronine (T3) and thyroxine (T4), iodinated amino acids, which are the active thyroid hormones. Diseases of the thyroid gland are manifested by qualitative or quantitative alterations in hormone secretion, enlargement of the thyroid, or both. Insufficient hormonal secretion results in hypothyroidism and, conversely, excessive secretion results in hyperthyroidism or thyrotoxicosis. Enlargement of the thyroid gland may be generalized or focal and may be associated with increased, normal,

* Resident in Family and Community Medicine, Sioux Falls, SD.

** Specialist in Internal Medicine, Sioux Valley Hospital; Assistant Clinical Professor, School of Medicine, University of South Dakota.

*** Specialist in Endocrinology, Sioux Valley Hospital; Assistant Clinical Professor of Medicine, School of Medicine, University of South Dakota.

**** Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Professor of Pathology, School of Medicine, University of South Dakota.

or decreased secretion, depending on the underlying disturbance.

The reactions involved in the synthesis and secretion of the active thyroid hormones can be divided into four sequential steps (Fig. 1).¹⁷ The first involves active inward transport of iodide from the plasma into the thyroid cell and follicular lumen. This "plasma iodide" is derived from two sources: iodine ingested in food, water, or medication and iodide derived either from the deiodination of thyroid hormones or from iodinated agents that the patient may have been given.

The second step in hormone biosynthesis results in the formation of hormonally inactive precursors, moniodotyrosine (MIT) and diiodotyrosine (DIT). These tyrosines are then "coupled", yielding a variety of iodothyronines, including T_3 and T_4 . These are stored as thyroglobulin, a glycoprotein that is synthesized within the follicular epithelium, and this serves as a storage form of thyroid hormone.

The third step in hormone synthesis and release involves hydrolysis of thyroglobulin, releasing the T_3 and T_4 , and the final release of the now-free iodothyronines (T_3 , T_4) into the blood, while the inactive iodothyronines (MIT, DIT) are stripped of their iodine by an intrathyroidal enzyme. It should be noted, however, that only a small proportion of

the T_3 in the blood is derived by direct thyroid secretion. The majority is derived from peripheral monodeiodination of T_4 .

In the blood, T_3 and T_4 are almost entirely bound to plasma proteins. T_4 is bound, in decreasing order of avidity to thyroxine-binding globulin (TBG), to a T_4 binding pre-albumin (TBPA), and to albumin, TBG being by far the major determinant of overall binding avidity. The interaction between T_4 and its binding protein conforms to a reversible binding equilibrium in which the majority of the hormone is bound and normally less than 0.5% is free. Because it is only the free hormone that is available to the tissues, the metabolic state of the patient correlates more closely with the concentration of free hormone rather than the total concentration of hormone in the plasma. Homeostatic regulation of thyroid function is directed toward maintenance of a normal concentration of free rather than total hormone.

Disturbances of the thyroid hormone-plasma protein interaction are of two types. The first results from a change in the concentration of TBG. For example, an increase in TBG causes an initial decrease in the free hormone concentration in the serum. As a consequence more hormone is released into the serum resulting in a return to normal concentration of the free hormone and an overall increase in total

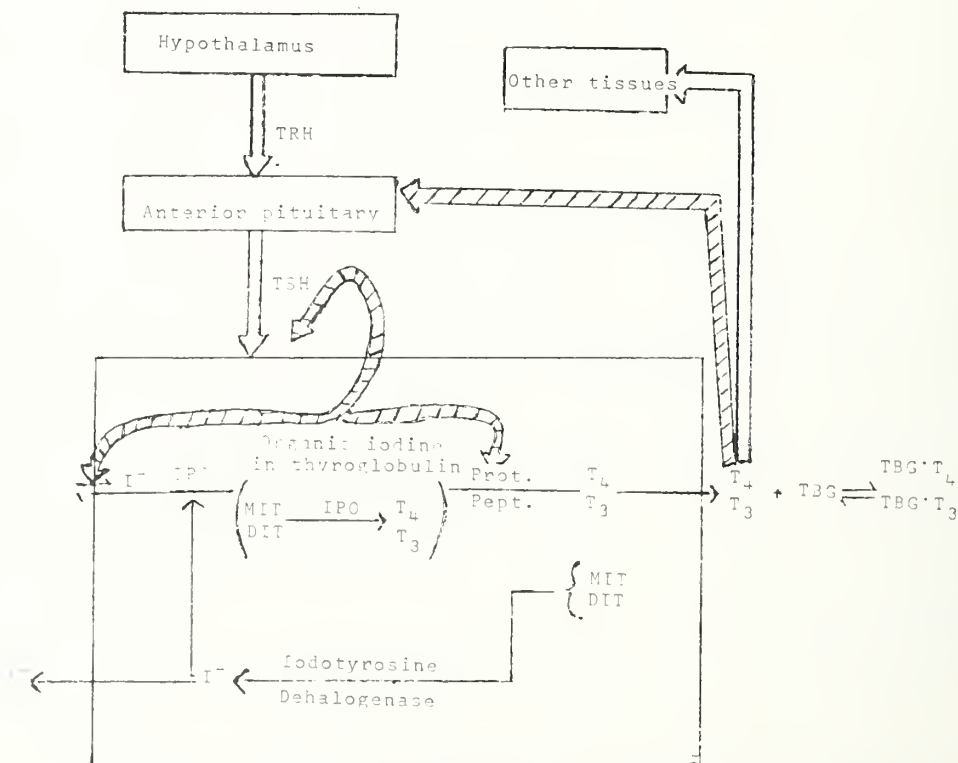


Figure 1

hormone concentration. Table I¹⁷ lists certain circumstances associated with altered concentration of TBG.

Table I ¹⁷	
Circumstances associated with altered concentration of TBG	
Increased TBG	Decreased TBG
Pregnancy	Androgenic and anabolic steroids
Oral contraceptives and other sources of estrogen	Large doses of glucocorticoid
Acute intermittent porphyria	Chronic liver disease
Chronic liver disease	Active acromegaly
Acute hepatitis	Nephrosis
Genetically determined	Genetically determined

The second type of disturbance results from a primary alteration in concentration of thyroid hormones in the blood such as occurs in hypothyroidism or thyrotoxicosis. Here the concentration of TBG is essentially unchanged and the concentration of free hormone will vary directly with the total concentration of hormone.

Thyroid function is regulated by two general mechanisms (Fig. 1).¹⁷ Thyroid-stimulating hormone (TSH) is secreted by basophilic cells in the anterior pituitary and stimulates thyroid hypertrophy and hyperplasia as well as all phases of thyroid function.

TSH secretion, in turn, is affected by two opposing influences. Thyrotropin-releasing hormone (TRH), secreted in the ventromedial hypothalamus, stimulates synthesis and secretion of TSH. These effects of TRH are inhibited, however, in the pituitary by a negative feedback system of thyroid hormones in the blood and this is presumed to be closely related to the concentration of free hormone in the blood.

With this brief review of thyroid physiology, I would like to now explain a few thyroid tests, what they measure, what they mean, and how they are helpful in diagnosing thyroid disease. Particularly, I will discuss those tests noted in today's case, and later, also mention a few other tests that would have been helpful in establishing a diagnosis for this patient.

At present, serum T_4 is measured by either a competitive protein-binding assay using TBG as the binding protein or by a radioimmunoassay using T_4 binding antibodies. The specificity of T_4 measurement by CPB and RIA is similar, but RIA is probably simpler to perform. Some feel it also is more sensitive although this may not necessarily be so. RIA does require less serum and can be performed with as little as 10 μ l of serum. Normal range for

T_4 by CPB is 4.0-11.0 μ l/100 ml and by RIA is 4.8-13 μ l/100 ml.

Serum T_4 is clearly elevated in more than 90% of patients with hyperthyroidism. It is normal, however, in some hyperthyroid patients who may have either T_3 thyrotoxicosis or a deficiency of circulating TBG. A deficiency of TBG may be expected in the nephrotic syndrome, hepatic cirrhosis, during treatment with androgens, and in persons who have a congenital defect in synthesis of TBG. On the other hand, when TBG is elevated, serum total T_4 will be high even in the absence of hyperthyroidism. TBG is elevated during pregnancy, estrogen therapy, infectious hepatitis, intermittent porphyria, and in a congenital disorder characterized by elevation of this serum protein.

Serum T_3 is measured by RIA. It is perhaps the single most sensitive test for diagnosis of hyperthyroidism. Nearly all hyperthyroid patients have a demonstrable increase in serum T_3 . Hyperthyroid patients have, in fact, been described in whom hypermetabolism can only be ascribed to elevated serum T_3 and serum T_4 is normal. Like T_4 , T_3 concentration is also influenced by serum concentration of TBG but probably less so.

Since abnormalities in the concentration of thyroid hormone binding proteins occur very commonly, it is prudent to include as assessment of free thyroid hormone levels in every initial examination of thyroid function. A test frequently used for this purpose is the T_3 resin uptake (RT_3U) which actually evaluates the degree of saturation of thyroid-binding proteins. By itself, it is not a good test of thyroid function; its main usefulness is adjustment of serum total T_4 for abnormalities in thyroid hormone binding proteins. In order to use the T_3 resin uptake test for "adjustment" of serum T_4 values, one divides the RT_3U in the patient by the mean normal RT_3U to obtain a ratio which is then multiplied by the patient's serum T_4 to obtain a free thyroxine index or "FTI". It is elevated when serum total T_4 is high because of hyperthyroidism and is normal when elevated T_4 is due to an increase in TBG.

The effective thyroxine ratio (ETR) gives the same information as the FTI and uses the patient's T_4 and TBG for measurement.

Thyrotropin (TSH) measurement is by RIA. Its level is normal or undetectable in pituitary or hypothalamic hypothyroidism and invariably increased in hypothyroidism of thyroidal origin. It is probably the best test to assess the presence of hypothyroidism.

The thyroid uptake (RAIU) involves oral administration of ^{131}I (more recently, ^{123}I) and then determining the amount of radioactivity accumulated by

the thyroid in 24 hours by counting over the thyroid location in the neck. In general, the RAIU is of relatively little usefulness in the diagnosis of the hyperthyroid state. Its chief value is in determining the cause of hyperthyroidism when the cause is in doubt, as will be shown later. The RAIU is typically high or high normal in hyperthyroidism due to Grave's disease or nodular goiter. It can also be high in iodine deficiency states and in patients taking diuretics. The RAIU is low, obviously, in patients whose thyroid is located outside its normal location since counting is done over the neck. It is also low in hyperthyroid patients whose hyperthyroidism is due to thyrotoxicosis factitia, subacute thyroiditis, a variant of chronic thyroiditis, or Hashimoto's disease. Patients with Grave's disease may also have a low RAU when they have ingested iodine, iodide or iodine-containing drugs.

The thyroid scan is intended mainly to evaluate the morphology and the relative functional status of the various portions of the gland. A thyroid scan is not necessary to determine that a hyperthyroid patient with a diffusely enlarged gland has Grave's disease.

Hyperthyroidism is confirmed by the use of in vitro thyroid function tests. The scan is useful, however, in a patient with a nodular goiter with thyrotoxicosis to assist in differential diagnosis since single or multiple hyperfunctioning nodules are readily apparent.

With this background in mind, I would like now to turn the discussion to today's case. Primarily I am going to discuss some causes of hyperthyroidism and hypothyroidism and how they might apply to this patient; I also want to discuss hypothyroidism in the postpartum state.

First of all, I would like to comment on the findings described by the obstetrician, three months prior to admission. On physical exam, the patient had a diffusely enlarged, non-tender thyroid, and had noted palpitations and her skin had become more oily. She had lost weight but had been dieting (this makes weight loss somewhat difficult to evaluate). On those observations alone, what can one say? Certainly all of the findings are suggestive of hyperthyroidism but all of them may also occur in a euthyroid pregnant woman. There is a normal hyperplasia of the thyroid in up to 70% of pregnancies. Because of this, hyperthyroidism is a more difficult disease to diagnose clinically in pregnant women. Problems in making the clinical diagnosis are compounded by changes in thyroid function tests during pregnancy which make the laboratory diagnosis difficult. Numbers are probably in our favor against hyperthyroidism in that thyrotoxicosis probably is less common in associa-

tion with pregnancy than it is in the general population. If she is indeed thyrotoxic, statistically it is most likely due to Grave's disease but it must be re-emphasized that most of the common signs and symptoms of hyperthyroidism also occur in a normal pregnancy.

What about her lab tests - normal T_4 and T_3 resin uptake (RT_3U)? A normal T_4 is seen in euthyroidism but a T_4 can also be normal in thyrotoxicosis. What about the normal T_3 resin uptake? Recall that the RT_3U does not measure T_3 but rather depends on the unsaturated binding capacity of TBG. With an increase in TBG in pregnancy, one would expect a RT_3U at or below the lower limits of normal for the euthyroid pregnant patient. Therefore, a normal RT_3U in pregnancy would be consistent with hyperthyroidism.

Is this patient then euthyroid or hyperthyroid? I do not know that we can say for sure with any great accuracy. Certainly, a FTI or ETR would be helpful in this circumstance to "adjust" for the increase in TBG.

What then about her initial findings on presentation to the internist in the postpartum period? Several physical findings certainly suggest a hyperthyroid state: her skin is oily and smooth, she has bilateral lid lag, the thyroid is enlarged, there is a soft flow murmur, fine tremor of the extremities, brisk DTR's - classic signs! Is she hyperthyroid at this point? Let's look at the laboratory values - T_4 is elevated, though not markedly; T_3 by RIA is certainly elevated, ETR is elevated. Yes, I would say she is hyperthyroid at this point. TSH is also reported and is normal, although I am not sure just why this was done, as TSH is not helpful in most cases of hyperthyroidism and, in fact, is probably not indicated when one begins a workup suspecting hyperthyroidism from clinical examination.

Let me just stop here a moment and consider a few possible diagnoses knowing just what we have discussed so far. I think we can say she is hyperthyroid. What's causing her to be such? What are the possibilities?

Grave's disease can certainly present with this picture. Classically, in Grave's disease there is the triad of hyperthyroidism with a diffuse goiter, ophthalmopathy and dermopathy. Although considered part of the same disease complex, the three major manifestations need not appear together and, in fact, one or two need never appear.

We can probably exclude toxic multinodular goiter or toxic adenoma because of the diffuse, symmetric thyroidal enlargement.

Self-administration of thyroid hormone, which is called thyrotoxicosis factitia, should always be kept

in mind and patients should be asked about this possibility. This patient's enlarged thyroid would rule out that diagnosis.

Ectopic thyroid, such as struma ovarii, is very rare but would need to be considered in a patient who is thyrotoxic without an enlarged gland.

Occasionally, tumors of trophoblastic origin may elaborate thyroid-stimulating substances. Very rarely, a TSH-secreting tumor of the pituitary could exist.

Subacute thyroiditis can cause transient hyperthyroidism but these patients usually have pain over the thyroid or pain referred to the lower jaw or ear. Recently there have been reports, though, of a "painless" subacute thyroiditis. This will be discussed again a little later.

Hashimoto's disease or autoimmune thyroiditis can first present with thyrotoxicosis.

Let's take into consideration now the thyroid uptake and scan. There was only 1% uptake in 24 hours and poor visualization of the thyroid. A very low RAIU in a patient who is thyrotoxic certainly limits our diagnoses.

Grave's disease may still fit but, generally, in this condition the RAIU would be normal or increased. About the only way this could be Grave's disease and fit this picture would be if the patient was getting excess iodine. There is no history of this, so we can probably eliminate Grave's disease from our possibilities.

Ectopic thyroid would, for technical reasons, give low uptake over the neck but, again, the enlarged thyroid probably eliminates this possibility.

We have a low TSH, so we know we are not dealing with a rare TSH-secreting tumor.

We are left with two possible diagnoses: 1) Painless subacute thyroiditis, or "silent" thyroiditis or 2) Autoimmune thyroiditis or Hashimoto's disease. These may appear similar clinically and, at this point, to differentiate them one may want to pursue further laboratory studies, namely, antithyroglobulin antibodies and anticrosomal antibodies. Needle biopsy may also prove useful. None of these were done in this patient. Typically, antithyroid antibodies are markedly positive in autoimmune thyroiditis but initially may also be positive in subacute thyroiditis. Some people feel that these two disorders may actually be just different parts of a spectrum of the same disease.

With either of these diagnoses, however, we can predict that the hyperthyroidism will be a transient phenomenon, as did this patient's internist. Therefore, he chose propranolol to treat her symptoms. Obviously, it would not be wise to put this patient on antithyroid drugs such as propylthiouracil or

methimazole and, in fact, antithyroid drugs are contraindicated in these conditions of transient hyperthyroidism.

The history then notes that in the ensuing months, now off all medicine, that she developed symptoms of hypothyroidism. Her laboratory tests confirm this. Note the decreased T_4 and elevated TSH. She was then treated with thyroid replacement and did well.

Returning to the previously mentioned possible diagnoses, what would give us this picture? Subacute thyroiditis is generally short-lived with a subsequent return to the euthyroid state. Hashimoto's disease could give us this picture; and, although the antithyroglobulin and antimicrosomal antibodies were not obtained in this patient, I would expect them to be present and markedly elevated and I have to suspect Hashimoto's disease as the diagnosis in this patient.

Hashimoto's disease is, as you know, the most common non-iatrogenic cause of hypothyroidism and is a chronic inflammatory disease of the thyroid in which autoimmune factors are thought to play a prominent role. It is a common disorder, occurring most frequently in women of middle age. Evidence of the participation of autoimmune factors includes the lymphocytic infiltration of the gland as well as the presence of increased concentration of immunoglobulins and of antibodies directed against several components of thyroid tissue. This disorder also co-exists with inordinate frequency with other diseases of a presumed autoimmune nature including pernicious anemia, Sjögrens syndrome, progressive hepatitis, systemic lupus erythematosus, rheumatoid arthritis, non-tuberculous Addison's disease and Grave's disease itself. These disorders, as well as Hashimoto's disease itself, also appear with unusual frequency in family members of patients with Hashimoto's disease.

I would like now to turn the discussion more specifically to hypothyroidism in the postpartum period. Several case reports very similar to this case have been in the recent literature, many showing initial transient thyrotoxicosis with subsequent, though again transient, hypothyroidism.¹²⁻¹⁸ In some studies this was felt to be due to a painless thyroiditis in the postpartum state,⁸ and in others, it is felt to be autoimmune thyroiditis.¹⁸

In 1976, in the *Journal of Clinical Endocrinology and Metabolism*, Amino *et al* in Japan reports the spontaneous occurrence of and recovery from primary hypothyroidism in six women with autoimmune thyroiditis. Diffuse goiter was noticed 1-3 months after delivery. The blood thyroid hormone level was found to be lowest at 3-6 months post-

partum. In 6-9 months after delivery, the enlarged thyroid gland decreased in size, and the values of thyroid function tests returned to the normal range without any treatment. Antithyroglobulin antibodies were positive in three cases. Antithyroid microsomal antibodies were positive in all cases with titers of 1:10⁴ to 1:10⁹. Titers were highest during the period of hypothyroidism and decreased thereafter. Although the enlarged thyroid gland decreased in size in all cases, the goiters did not completely disappear during observation periods of 6 months to 6 years but the consistency changed from soft to elastic and firm. Although it is generally agreed that hypothyroid patients with chronic thyroiditis should be treated with thyroid hormone throughout life, from the findings in these cases, it does not seem necessary to treat patients who develop primary hypothyroidism after delivery. If TSH is important for recovery of thyroid function as some have suggested, thyroid replacement therapy should perhaps not be used in such cases.

In *Lancet*, May 28, 1977, Ginsberg and Walfish reported five patients who presented with transient thyrotoxicosis and painless thyroiditis in the postpartum period. Thyrotoxicosis developed within 1-6 months of delivery. All had small non-tender goiters. Initially all had elevated values for T₄ and T₃ by RIA. RAIU was suppressed in all. In all patients, thyrotoxicosis resolved within four months, subsequent transient hypothyroidism occurred in 4, 1 developing permanent myxedema. Two had persistently elevated thyroid antibody titers and needle biopsy findings compatible with chronic thyroiditis. These authors felt that since the disorder is self-limited, conservative therapy should be given. They suggested following thyroid antibody titers and note that if they remain elevated, this person is probably more likely to remain hypothyroid.

In the *Annals of Internal Medicine* in 1977, Amino et al report 25 episodes of postpartum primary hypothyroidism observed in 23 patients without hormone treatment. Three were cases of irreversible hypothyroidism and the others were of transient hypothyroidism. The characteristics of transient postpartum hypothyroidism deduced by serial observations of 14 patients are noted in Table II. Transient postpartum hyperthyroidism before the occurrence of hypothyroidism were observed during two consecutive postpartum periods in two patients. Transient hypothyroidism was also observed in two patients after abortion. Fourteen of the 23 patients first noted thyroid abnormality after delivery.

These findings support the idea that parturition is associated with the onset of overt autoimmune thyroiditis. These authors also suggest that transient

hypothyroidism following transient hyperthyroidism may not be a rare phenomenon in patients with autoimmune thyroiditis. In patients with Grave's disease and those with autoimmune thyroiditis, postpartum transient hyperthyroidism was observed less than three months postpartum and transient hypothyroidism developed 3 to 4 months postpartum. These findings suggest that similar factors may induce these symptoms in both diseases.

Table II

1. A high incidence of previous goiter
2. Thyroid enlargement at ½ to 4 months postpartum
3. Hypothyroidism at 3 to 5 months postpartum
4. Spontaneous recovery at 5 to 10 months postpartum
5. High titers of antithyroid microsomal antibodies
6. Persistence of small goiter

As mentioned, these phenomena have also been noted in a patient with Grave's disease who had been in remission due to antithyroid therapy, but I do not feel that today's patient has Grave's disease for reasons previously mentioned.

It has recently been suggested that immune reactions are suppressed to some degree in pregnancy. In other autoimmune diseases such as rheumatoid arthritis, systemic lupus erythematosus, and idiopathic thrombocytopenic purpura, relief of symptoms was often observed during pregnancy with relapse or even aggravation of the disease 2 to 6 months postpartum. These authors think that the phenomenon of transient hypothyroidism may be induced by the immunologic change occurring after delivery during the course of autoimmune thyroiditis, but further studies are necessary to clarify its exact cause.

I would like to open the discussion at this point in regards to the treatment of this patient with L-thyroxine. Certainly she is hypothyroid and replacement therapy is indicated in this final stage of Hashimoto's disease, but, in the light of these current studies, might this just be a transient phenomenon in this patient as it was in the cases reported in the literature?

Dr. Tom Grau's Diagnoses

Hashimoto's or Autoimmune Thyroiditis

DR. ASPAAS: Certainly the patient had transient hyperthyroidism followed by severe hypothyroidism. She may not require treatment forever. When the patient came into my office the second time, she was highly symptomatic with signs of hypothyroidism and had a T₄ level of 1.4 ug/dl. There was no way I could have avoided giving this lady thyroid hormone to relieve her symptomatology. There were several things that make me suspect that this patient

had a so-called autoimmune thyroiditis or Hashimoto's thyroiditis rather than subacute or viral thyroiditis which is often characterized by transient hyperthyroidism followed by transient hypothyroidism with recovery. One of these factors was the time frame in that she did not become hypothyroid until 6-7 months after her episode of hyperthyroidism. This would seem to be a little long for most cases of subacute thyroiditis. Another point is that she had very dramatic hypothyroidism rather than the mild hypothyroidism one might see if the disease were going to be of a self-limited nature. I would ask Dr. Lovrien whether I should try to taper the patient off thyroid hormone to see if she would spontaneously revert to the euthyroid state.

DR. LOVRIEN: It is not known whether the use of thyroid hormone in cases such as this will lessen the chances of spontaneous recovery by the thyroid gland. There are also too few case reports to assess whether this patient will remain permanently hypothyroid or can revert to the euthyroid state. I believe that if the patient were taken off the thyroid hormone medication; she would, at least, go through a transient period of hypothyroidism, but you might have to wait two or three months to see if the patient's thyroid gland would recover. I do not know a way of predicting recovery in these patients.

DR. ASPAAS: Is there any correlation with the severity of the hypothyroidism and the prognosis for recovery?

DR. LOVRIEN: Some reports have suggested that if the T_4 level is below 1, permanent hypothyroidism is more likely, but I am not sure that there is enough data on this to make definite statements.

I think you always have to ask yourself whether it is worse having the patient go through 2-3 months of hypothyroidism to see if her gland will recover.

*DR. STASSEN: Would it be worth measuring her antithyroid antibody titers to determine whether her gland will recover?

DR. LOVRIEN: I think if the titers were high it would be less likely that she would become euthyroid.

DR. ASPAAS: My goal was to relieve the patient of hypothyroidism and decrease the size of her enlarging goiter. I probably will plan to keep her on thyroid medication indefinitely.

**DR. LLOYD SWEENEY: Can nuclear medicine give you any way of distinguishing between primary thyroid atrophy and other causes of hypothyroidism such as thyroiditis?

DR. BARLOW: I think if one uses the nuclear medicine tests in conjunction with other data one can usually determine this. A patient who has hypothyroidism with a goiter does not have primary thyroid atrophy. If the patient has a high level of antithyroid antibodies to either thyroglobulin or microsomal antigen in conjunction with goiter, the chances are that the patient has autoimmune thyroiditis. If the patient has low thyroid antibody titers with goiter, the patient may have painless subacute thyroiditis. DR. LOVRIEN: It must be pointed out, however, that low levels of thyroid antibodies may be seen in autoimmune thyroiditis. High levels are of significant value but low levels give us very little help. We know that low levels are more common in subacute thyroiditis but biopsy may prove the patient really has autoimmune thyroiditis.

DR. GRAU: I should like to point out that my questions concerning the possibilities of spontaneous recovery of the gland are academic. There is no doubt that this patient was hypothyroid and should have been treated. This is certainly what I would have done. I should also have mentioned for completeness that in the postpartum state, hypothyroidism may be due to pituitary necrosis (Sheehan's syndrome); but this is not a possibility in this case since the patient had a high TSH.

***DR. WILLIAM O. ROSSING: Are there advantages of measuring the T_4 by one method or another?

DR. BARLOW: The T_4 by radioimmunoassay is a much more sensitive and accurate test and is the most preferred one at this time as compared to the method using comparative protein binding with thyroglobulin as the binder. The radioimmunoassay uses a specific antibody and it is preferred.

A PHYSICIAN: What about the effective thyroxine ratio (ETR)?

DR. BARLOW: The ETR can be used as a general screen for thyroid function and is not altered by changes in the thyroid binding globulin which tend to depress or elevate the T_4 value. This has been explained by Dr. Grau. My own feeling is that if the patient is suspected of having hyperthyroidism, I would select the T_4 and T_3 both performed by radioimmunoassay as the tests of most value since one or both of these tests should be elevated in a patient who is thyrotoxic. On the other hand, if I were suspecting hypothyroidism, I would obtain T_4 and a TSH level, both performed by radioimmunoassay. If the T_4 is low and the TSH is elevated, cer-

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tainly a diagnosis of primary hypothyroidism on the basis of an inadequately functioning thyroid gland can be made. If the T_4 is low and the TSH is not elevated, one must consider a pituitary or hypothalamic origin as a possibility for the hypothyroidism.

DR. LOVRIEN: I would agree. However, I use the T_4 as my first line screening test. I would like to emphasize that a major problem is the interpretation of the T_4 or ETR when borderline values are obtained. When grossly abnormally depressed or elevated values are obtained and the clinical picture fits, there is little problem. However, minor degrees of elevation or depression of these tests do not lead to an automatic diagnosis and the patient should be further evaluated.

DR. BARLOW: I would like to just re-emphasize one point and this concerns the use of the radioactive iodine uptake test (RAIU). This is not a particularly good test for the evaluation of thyroid function due to frequently depressed values because of the presence of high amounts of iodides in diagnostic X-rays and the foods we eat. However, this test is very valuable in evaluating the patient with thyrotoxicosis. In the most common causes of thyrotoxicosis, Grave's disease, with diffuse hyperplasia and single or multiple hyperfunctioning nodules of the thyroid as well as the rare case of a pituitary neoplasm secreting TSH, the RAIU is elevated. However, there are several causes of hyperthyroidism where the RAIU is paradoxically depressed. One of the most common is a form of thyroiditis which is usually associated with self-limited hyperthyroidism followed by self-limited or permanent hypothyroidism. These patients may constitute 4%-16% of the cases of thyrotoxicosis as described by Woolf.⁸ Dr. Grau has pointed out these patients do not need ablative radioiodine or surgical therapy or antithyroid medications. Propranolol will be adequate for their transient thyrotoxic phase. In surreptitious ingestion of thyroid hormone (thyrotoxicosis factitia), there is no goiter and there is a prompt rise in the RAIU when the gland is stimulated by TRH. This is not true in thyroiditis.

Other rare causes of thyrotoxicosis include hyperfunctioning struma ovarii and metastatic hyperfunctioning thyroid cancer. The former can be detected on scanning the pelvis after injection of radioiodine and the latter can be diagnosed by history and appropriate scanning. Last but not least, a patient may have thyrotoxicosis after the ingestion of massive doses of iodides. This type of hyperthyroidism has been labeled with the eponym Jod-Basedow's disease. A history of ingestion of large doses of iodides has to be obtained to make this diagnosis.

In summary, since thyrotoxicosis can be due to many different causes and the treatment could vary markedly depending on the cause, the RAIU is a helpful test in making the proper distinctions.

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For use in the treatment of infant colic (syrup).

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy), obstructive disease of the gastrointestinal tract (as in achalasia, pyloro-duodenal stenosis), paralytic ileus, intestinal atony of the elderly or debilitated patient; unstable cardiovascular status in acute hemorrhage; severe ulcerative colitis; toxic megacolon complicating ulcerative colitis; myasthenia gravis. **WARNINGS:** In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. **PRECAUTIONS:** Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy. Use with caution in patients with: Autonomic neuropathy. Hepatic or renal disease. Ulcerative colitis. Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon. Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension. Hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.

Do not rely on the use of the drug in the presence of complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. **ADVERSE REACTIONS:** Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia; urinary hesitancy and retention; blurred vision and tachycardia; palpitations; mydriasis; cycloplegia; increased ocular tension; loss of taste; headache; nervousness; drowsiness; weakness; dizziness; insomnia; nausea; vomiting; impotence; suppression of lactation; constipation; bloated feeling; severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating. With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. **DOSEAGE AND ADMINISTRATION:** Dosage must be adjusted to individual patient's needs.

Usual Dosage: Bentyl 10 mg. capsule and syrup: *Adults:* 1 or 2 capsules or teaspoonfuls syrup three or four times daily. *Children:* 1 capsule or teaspoonful syrup three or four times daily. *Infants:* ½ teaspoonful syrup three or four times daily. (May be diluted with equal volume of water.) Bentyl 20 mg.: *Adults:* 1 tablet three or four times daily. Bentyl Injection: *Adults:* 2 ml. (20 mg.) every four to six hours intramuscularly only. NOT FOR INTRAVENOUS USE. **MANAGEMENT OF OVERDOSE:** The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal. Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested. If indicated, parenteral cholinergic agents such as Urecholine[®] (bethanechol chloride USP) should be used.

Product Information as of October, 1978.

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Letters To The Editor

I am writing this letter to give my sincere thank you to you and the State Medical Association in presenting me with a \$500 award. I am currently a freshman student and will find the money very helpful in my continued education at the USD School of Medicine, especially due to the rising cost of everything!

Again, my sincere thanks.

Respectfully yours,
Mark Mogen

I would like to thank you and the South Dakota State Medical Association for the generous scholarship which I recently received. The money is heartily appreciated and will certainly help me meet my financial needs. Thanks again for the scholarship.

Sincerely,
Dave Johnson

When I received the letter of notification for the South Dakota State Medical Association Award, I was very pleased and honored. This award came at a most opportune time and helped me and my family tremendously. Your consideration and support of the medical students of South Dakota is truly appreciated.

Thank you so much for your Association making this award available to myself and other medical students. The South Dakota State Medical Association is to be applauded.

Sincerely yours,
Wayne E. Snyder

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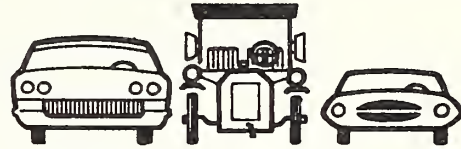
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Council Meeting Highlights

The Council of the South Dakota State Medical Association met on Friday and Saturday, April 27, 28, 1979, at the Ramada Inn, Sioux Falls, South Dakota. The following are major items of business transacted at this meeting.

1. **AMA RURAL HEALTH CONFERENCE.** The Council voted to reimburse expenses, up to \$350, for a physician to attend the 1980 AMA Rural Health Conference.

2. **EARLY AND PERIODIC SCREENING, DIAGNOSIS AND TREATMENT (EPSDT) REPORT/CLAIM FORMS.** The Council strongly endorsed the concept requiring physician signatures on EPSDT forms.

3. **NATIONAL HEALTH SERVICE AND RURAL HEALTH INITIATIVE PROGRAMS.** The Council recommended that the Commission on Medical Service meet periodically with the National Health Service and Rural Health Initiative program representatives to discuss what effect these programs have on existing medical practices in the area.

4. **STATEMENT ON HOME DELIVERIES.** The Council adopted the following statement as Association policy:

"Labor and delivery, while a physiologic process, clearly presents potential hazards to both mother and fetus before and after birth. These hazards require standards of safety which are provided in the hospital setting and cannot be matched in the home situations.

We recognize, however, the legitimacy of the concern of many that the events surrounding birth be an emotionally satisfying experience for the family. The Association supports those actions that improve the experience of the family while continuing to provide the mother and her infant with accepted standards of safety available only in the hospital."

5. **DRUG LAW.** The Council recommended that representatives of the SDSMA, the Health Department, the Pharmaceutical Association and the Attorney General's office review the entire drug law and specifically the problem with Class 2 prescriptions which requires a physician's signature within 72 hours of receipt of the telephoned prescription.

6. **BYLAW CHANGE ON CONTINUING MEDICAL EDUCATION REQUIREMENT.** The Council will propose to the House of Delegates a bylaw change which calls for a two year moratorium on the CME requirement while further study and specific methodology can be established for the SDSMA program.

7. **REQUEST FROM BOARD OF NURSING TO ENDORSE CONTINUING EDUCATION REQUIREMENT FOR NURSES TO MAINTAIN THEIR LICENSES.** The Council stated that although the SDSMA favors continuing education for allied health professionals, mandatory continuing education for nurses should be studied further and consideration should be given to the existing local quality education programs available through hospitals and other employers of nurses.

8. **AUXILIARY PARTICIPATION IN SDSMA ACTIVITIES.** The Council voted to invite the Auxiliary to name representatives to attend the meetings of the Commission on Internal Affairs, Communications and Liaison and the Commission on Legislation and Governmental Relations.

9. **PROPOSAL FROM AVIS RENT A CAR.** The Council accepted the proposal from Avis Rent A Car which would allow a discount for car rentals through Avis for members of the SDSMA.

10. **APPOINTMENT TO SODAPAC BOARD OF DIRECTORS.** Mrs. Sandy Swanson, Pierre, was appointed to the SoDaPac Board of Directors.

11. **APPOINTMENT TO ENDOWMENT BOARD OF DIRECTORS.** The Council reappointed the Endowment Board of Directors to serve a one year term. Members include: Doctors G. E. Tracy, Warren Jones, R. R. Giebink, Joseph Hamm, James Ryan, Anthony Javurek, and Durward Lang.

12. **IMPAIRED PHYSICIAN PROGRAM.** The Council adopted the proposed Impaired Physician Program for the state of South Dakota. ■

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INDICATIONS: *Therapeutically*, (as an adjunct to systemic therapy when indicated), for topical infections, primary or secondary, due to susceptible organisms, as in: infected burns, skin grafts, surgical incisions, otitis externa; primary pyodermas (impetigo, ecthyma, sycosis vulgaris, paronychia); secondarily infected dermatoses (eczema, herpes, and seborrheic dermatitis); traumatic lesions, inflamed or suppurating as a result of bacterial infection. *Prophylactically*, the

ointment may be used to prevent bacterial contamination in burns, skin grafts, incisions, and other clean lesions. For abrasions, minor cuts and wounds accidentally incurred, its use may prevent the development of infection and permit wound healing.

CONTRAINDICATIONS: This product is contraindicated in those individuals who have shown hypersensitivity to any of its components. Do not use in the eyes or in the external ear canal if the eardrum is perforated.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control

secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

SD

Future Meetings

July

Joint Meeting of American Society of Clinical Pathologists and College of American Pathologists, Washington, DC, July 7-14. Contact: American Society of Clinical Pathologists, 2100 W. Harrison St., Chicago, IL 60612.

Topics in Emergency Medicine, Colby College, Waterville, ME, July 8-12. 24 hrs. AMA Category I credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby Coll., Waterville, ME 04901.

Surgical Techniques, Colby College, Waterville, ME, July 17-20. 15 hrs. AMA Category I credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby Coll., Waterville, ME 04901.

Coronary Disease, Exercise Testing, and Cardiac Rehabilitation, Playboy Resort, Lake Geneva, WI, July 27-29. Fee: \$202. 13 hrs. AMA Category I credits. Contact: International Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

August

Ophthalmology, Colby College, Waterville, ME, Aug. 5-9. AMA and AAFP credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby College, Waterville, ME 04901.

Black Hills Summer Seminar, Holiday Inn of the Northern Hills, Spearfish, SD, Aug. 9-11. AMA and AAFP credits. Contact: Black Hills Summer Seminar, c/o SDSMA, 608 West Ave., N., Sioux Falls, SD 57104.

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, Four Seasons, Lake of the Ozarks, MO, Aug. 10-12. 13 hrs. AMA Category I credits. Fee: \$215. Contact: Internat'l. Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Obstetrics and Gynecology, Colby College, Waterville, ME, Aug. 12-16. AMA and AAFP credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby College, Waterville, ME 04901.

Nuclear Medicine, Colby College, Waterville, ME, Aug. 12-17. AMA and AAFP credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby College, Waterville, ME 04901.

Medical Staff Law and Bylaws, The Abbey on Lake Geneva, Lake Geneva, WI, Aug. 13-15. Fee: \$350. Contact: Registrar, Aspen Systems Corp., 20010 Century Blvd., Germantown, MD 20767

Great Debates in Otolaryngology, Washington Plaza Hotel, Seattle, WA, Aug. 16-18. Fee: \$285. 23 hrs. AMA Category I credits. Contact: CME, U. of Wash. School of Medicine, E-303 HSB, SC-50, Seattle, WA 98195.

Coronary Disease, Exercise Testing and Cardiac Rehabilitation, Dunfey's Lodge, Cape Cod, MA, Aug. 17-19. Fee: \$202. 13 hrs. AMA Category I credits. Contact: Internat'l Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, Inn on the Park, Toronto, Canada, Aug. 17-19. Fee: \$215. 13 hrs. AMA Category I credits. Contact: Internat'l Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Forensic Medicine, Colby College, Waterville, ME, Aug. 19-23. AMA and AAFP credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby College, Waterville, ME 04901.

Pulmonary Disease, Colby College, Waterville, ME, Aug. 21-23. AMA and AAFP credits. Contact: R. H. Kany, Dir., Div. of Special Programs, Colby College, Waterville, ME 04901.

Mackinac Island Conference, The Grand Hotel, Mackinac Island, MI, Aug. 24-27. AMA Category I credits. Contact: Office of CE, Towsley Center for CME, U. of Michigan Med. School, Ann Arbor, MI 48109.

24th Annual Conference, American Institute of Ultrasound in Medicine, Montreal, Canada, Aug. 27-31. Contact: Am. Inst. of Ultrasound in

Medicine, 6161 N. May, Suite 278, Oklahoma City, OK 73112.

September

American Cancer Society, National Conference—Breast Cancer 1979, Waldorf-Astoria Hotel, New York, NY, Sept. 6-8. 16 hrs. AMA Category I credits. Contact: Breast Cancer Conf., Am. Cancer Soc., 777 Third Ave., New York, NY 10017.

First National Conference on Antibiotic Review: West Coast Update, Bonaventure Hotel, Los Angeles, CA, Sept. 10-11. Contact: Muriel Myers, Suite 113, 67 Peachtree Park Dr., Atlanta, GA 30309.

Seminar for Family Physicians, U. of Vienna, Vienna, Austria, Sept. 17-21. Contact: M. Arthur Kline, M.D., American Med. Soc. of Vienna, Lazarettgasse 13, Vienna IX, Austria.

Coronary Disease, Exercise Testing and Cardiac Rehabilitation, Sheraton on the Wharf, San Francisco, CA, Sept. 28-30. Fee: \$202. 13 hrs. AMA Category I credits. Contact: Internat'l Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, Sahara Hotel, Las Vegas, NV, Sept. 28-30. Fee: \$215. 13 hrs. AMA Category I credits. Contact: Internat'l Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

October

Seminar in Internal Medicine, U. of Vienna, Vienna, Austria, Oct. 1-5. Contact: M. Arthur Kline, M.D., American Med. Soc., of Vienna, Lazarettgasse 13, Vienna IX, Austria.

Fourth Annual International Body Imaging Conference, Royal Lahaina Hotel, Maui, HI, Oct. 13-21. Fee: \$295. 30 hrs. AMA Category I credits. Contact: Conf. Coord., Fourth Ann. Body Imaging Conf., West Park Hosp., Dept. of Radiology, 22141 Roscoe Blvd., Canoga Park, CA 91304.

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Table of Contents: page 3

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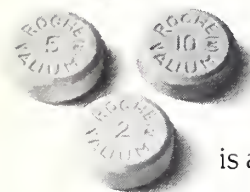


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Another important aspect of the clinical character of Valium is safety. Though drowsiness, ataxia and fatigue are possible, these and more serious side effects are rarely a problem. Of course, as with all CNS-acting drugs, patients taking Valium should be cautioned against driving, operating dangerous machinery or the simultaneous ingestion of alcohol.

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The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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SCIENTIFIC ARTICLES

- 7 Two Unusual Scrotal Masses in Young Boys
John H. Hoskins, M.D.
John F. Barlow, M.D.

- 14 X-Ray Case of the Month
Martin F. Petereit, M.D.
Donald J. Peik, M.D.

FEATURES

- 21 President's Page
23 South Dakota AFP Chapter News
24 Laboratory Aids
32 Future Meetings

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Two Unusual Scrotal Masses in Young Boys

John H. Hoskins, M.D.*

John F. Barlow, M.D.* *

ABSTRACT

A case of leiomyoma adjacent to the testicle and a case of intrascrotal accessory spleen are reported to illustrate that benign solid tumors in or near the testis must be considered in the differential diagnosis of scrotal swellings. Exploration of the scrotum and testis can result in resection of the tumor and salvage of the testis on some occasions. The intrascrotal spleen represents an example of splenic-gonadal fusion which is briefly reviewed.

INTRODUCTION

An unusual leiomyoma of the testis was situated below a prepubertal testis of normal histology of a twelve year old boy. The neoplasm was pedunculated and had undergone torsion but had not given rise to any symptoms severe enough to demand medical attention. The case is reported because of its unusual nature and location. It was felt not to be a leiomyoma of the epididymis or a paratesticular fibrous pseudotumor previously described.¹⁻⁴

The other intrascrotal mass in a fifteen year old boy is one form of splenic gonadal fusion exemplified by paratesticular normal accessory splenic tissue. This entity is always on the left side. Other examples of splenic gonadal fusion can have con-

tinuous or discontinuous fragments of splenic tissue along a path from the location of the normal spleen to the scrotum. The continuous form is associated with ectomelia.^{5,6}

Case No. 1 - 717212

This 12-year old boy was admitted to Sioux Valley Hospital because of enlarged right testis had been noted on a physical examination prior to entrance to school. The patient had no history of pain or trauma. The past medical history and review of systems were unremarkable. The physical examination was within normal limits except that the right testicle was felt to be three times the size of the left, very hard, nodular and nontender. The left testicle was descended and normal to palpation. Chest x-ray was negative. Urinalysis, hemoglobin, and white count were within normal limits. Pregnancy test was negative. The patient underwent inguinal exploration. The testicle was delivered, inspected and high ligation of the spermatic cord with removal of the cord and right testis was carried out. On pathologic examination the testis was $2.5 \times 1.5 \times 1.8$ cm. attached to a normal spermatic cord. Attached to the testis inferiorly in a pedunculated fashion was a $4.0 \times 2.8 \times 2.0$ cm very discrete firm mass which on cut section had a solid bulging tan surface with traversing broad bands of white fibrous tissue. There was no cyst formation or hemorrhage. (Figs. 1 & 2)

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** Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital, Sioux Falls, SD; Professor of Pathology, School of Medicine, University of South Dakota.

Microscopic examination revealed a normal prepubertal testis. The adjacent tumor was composed of fasciculi of spindle cells cut in various directions. Cell borders were not clear and there was a small amount of reddish cytoplasm. The lesion had focal increase in cellularity and there were rare mitoses. An actual mitoses count was 2 per 50 high power fields in one area but most areas of the tumor showed no mitotic activity. There was no atypicality to the nuclei which were vesicular with blunted ends. Nucleoli were inconspicuous. Masson trichrome stain suggested fibrous and smooth muscle elements and was similar to a leiomyoma of the uterus. (Figs. 3 & 4)

Post operatively the patient had no complications and returned to normal activity.



Figure 1
Pedunculated leiomyoma beneath testis.



Figure 2
Cut section of leiomyoma on twisted stalk below testis immediately above.



Figure 3
Low power of vascular spindle cell leiomyoma.



Figure 4
High power to show blunt end nuclei of leiomyoma.

Case No. 2 - 746298

This 15-year old boy had a left testicular asymptomatic mass noted on physical examination. He had no pain, trauma, or symptoms referable to the genitalia. The patient's general health had been excellent and there was no history of hospitalization or serious illness. Review of systems and past history were unremarkable. The boy was an alert 15 year old, 142 lb. male, in no distress. Vital signs were within normal limits. The right testis was normal in size and normal to palpation. The left testis felt normal but there was a contiguous mass above the left testis which was very firm and smooth. The tumor did not transilluminate. There were no other abnormalities on physical examination. Exploration through an inguinal incision revealed a mass above the left testis. Biopsy was sent for frozen section and the report was benign lymphoid tissue. The mass was excised and submitted. The mass was a 2 cm in diameter homogenous red structure which was dark red and uniform on cut section. (Fig 5)

Permanent microscopic sections showed normal splenic tissue with red pulp and white pulp. (Fig. 6) There was a

typical capsule about the splenic tissue. Tubules outside the capsule showed hypospermatogenesis.

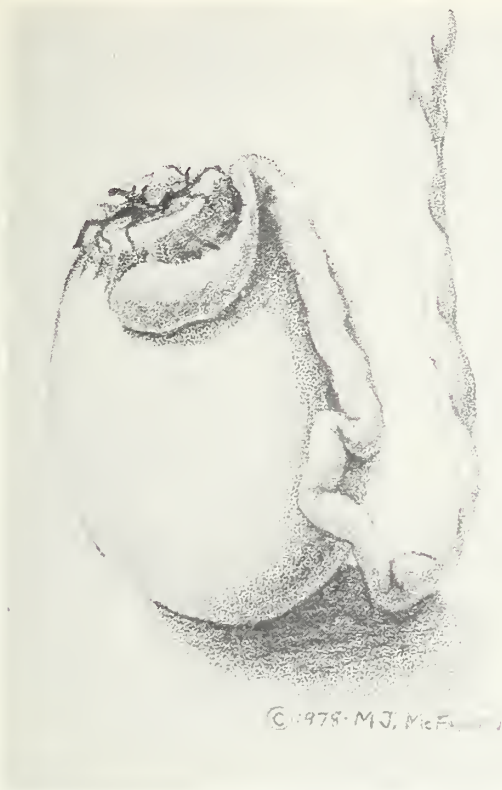


Figure 5

Drawing illustrating splenic nodule on top of testis. Crescent shaped structure below splenic nodule represents epididymis.

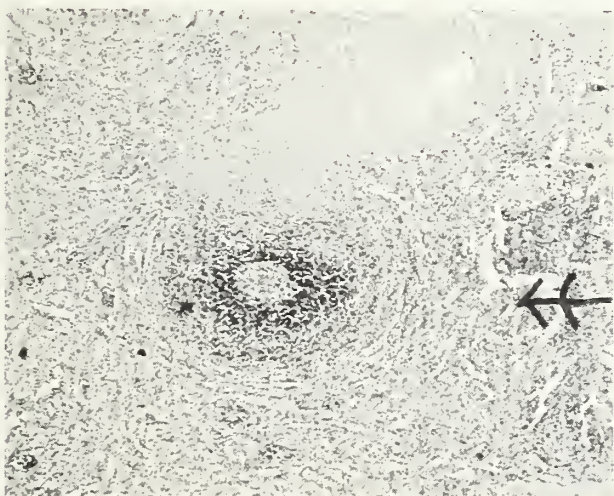


Figure 6

Typical splenic tissue, arrows point to central white pulp surrounded by red pulp.

COMMENT

Intrascrotal masses in adolescents are not usual and can lead to confusion in preoperative evaluation. Reported is a twisted paratesticular benign

spindle cell tumor situated below the testis which was displaced cephalad by the tumor. The neoplasm was preoperatively easily confused with an infarcted testis which had undergone torsion or a possible testicular neoplasm. The spindle cell tumor had areas of fibrous tissue but there were also definite elements which were histologically similar to a leiomyoma of the uterus. The fibrous tissue portion may have been secondary to ischemic changes secondary to impairment of blood supply from torsion of the stalk of the tumor. The neoplasm was not connected to the epididymis and did not fit the description of the reactive fibrous pseudotumors described by Mostofi and others.¹⁻⁴

The occurrence of splenic tissue elsewhere than in the primary organ is intriguing. The spleen develops in the dorsal mesogastrium and is recognized about the fourth or fifth week of embryonic life as multiple masses of splenic tissue which fuse to form the spleen. This fusion explains the notches seen in the normal spleen. Tissue from this splenic anlagen can be carried to other areas as the dorsal mesogastrium is converted into various peritoneal structures. Accessory spleens thus may be seen in the hilus of spleen, splenic pedicle, gastric ligaments and mesenteries, retroperitoneum near the tail of the pancreas, omentum and mesenteries of small and large bowel.⁷ Accessory spleens can undergo torsion, be seen as masses indenting the gastrointestinal outline on x-ray, or produce recurrence of primary splenic conditions such as hereditary spherocytosis or immune thrombocytopenic purpura after splenectomy.^{7,8} Accessory spleens should be distinguished from splenosis which is the implantation of multiple fragments of spleen into the peritoneal cavity after trauma.⁷

Of particular pertinence to this case is that the fusing precursors of the spleen are in close contact with the dorsal genital ridge during embryologic development. Fusion of the genital and splenic embryonic structures may produce splenic tissue anywhere from the left upper quadrant to the scrotum when the gonad descends about the eighth embryologic week.

Sixty-six cases of splenic-gonadal fusion have been reported, mostly in caucasians.⁵ Thirty-seven cases were of the continuous variety in which cords of splenic or fibrous tissue ran from the spleen to the scrotum anteriorly or laterally by a transperitoneal route, or rarely retroperitoneally. In twenty-nine cases the splenic-gonadal fusion was discontinuous or separated from the primary spleen.⁵ This anomaly has often been found at autopsy, but splenic tissue as an incidental finding at operation

for herniorrhaphy or cryptorchidism has occurred. Preoperative confusion with testicular neoplasms, epididymitis, epididymal tumors or supernumerary testis when the spleen presents as a scrotal mass certainly suggest that accessory scrotal splenic tissue be included in the differential diagnosis of intra-scrotal lesions.⁵⁻¹⁴ Consideration of salvage of the testis must be entertained at exploration. The presence of left-sided scrotal mass attached to the tunica albuginea should enhance the suspicion of splenic-gonadal fusion.

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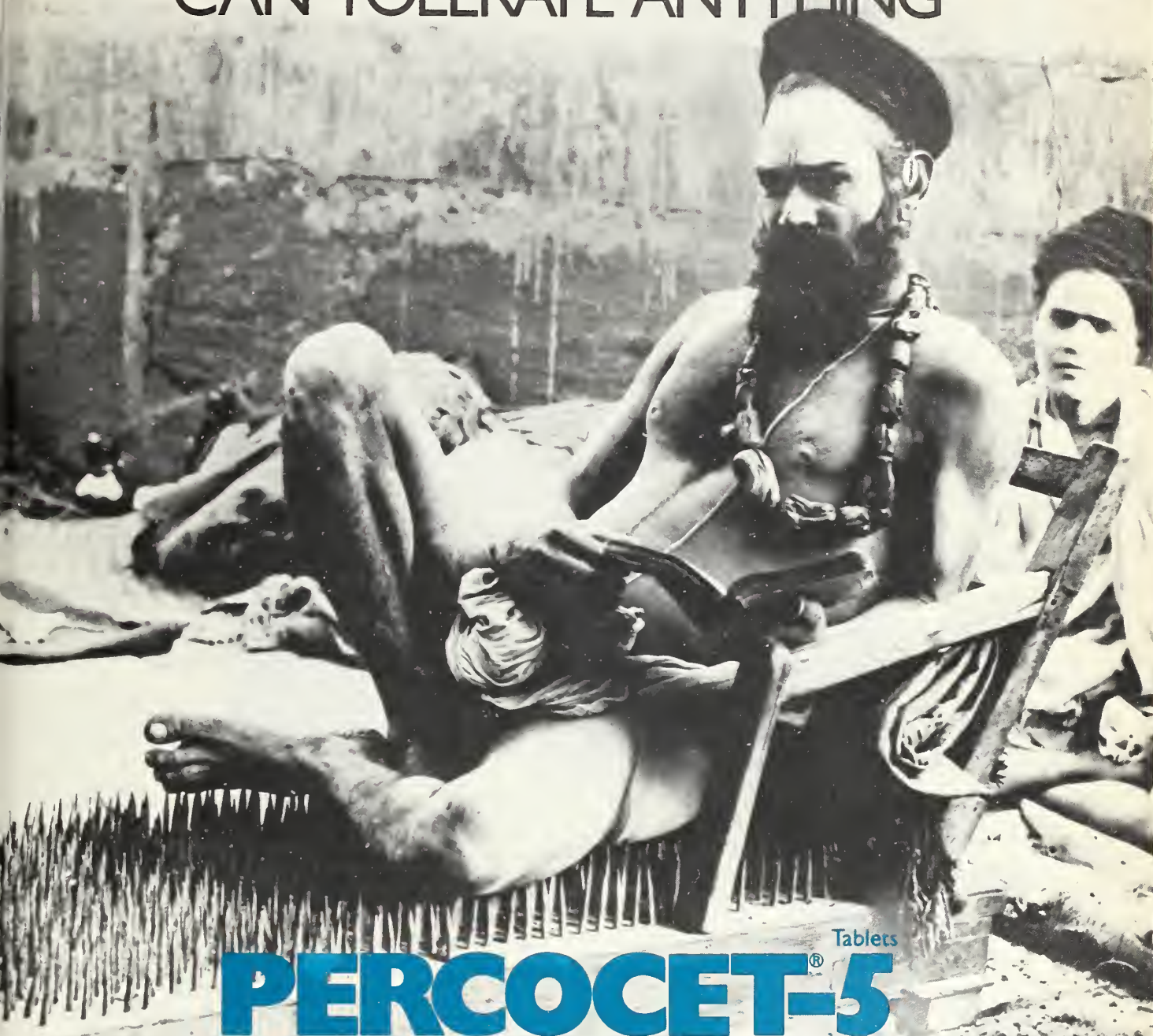
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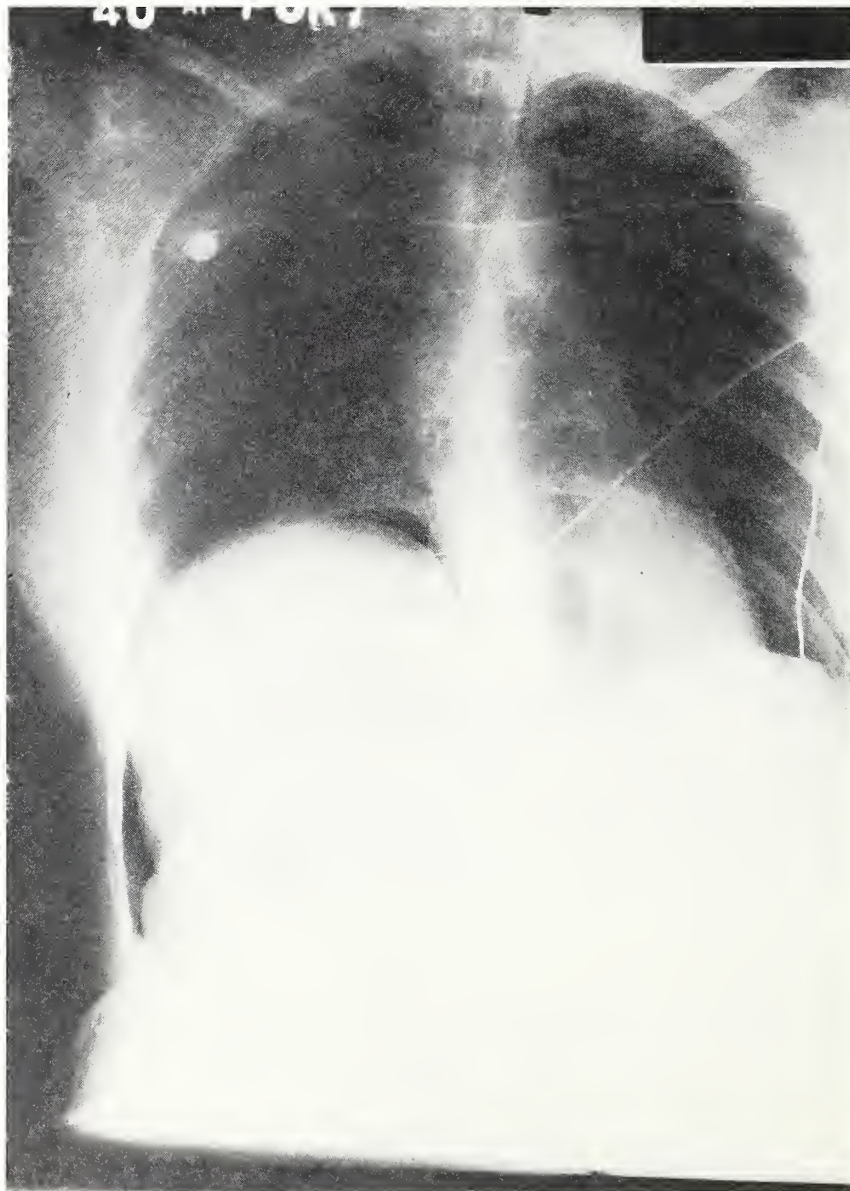


Figure 1

* Radiologists, Medical X-ray Center, Sioux Falls, SD.

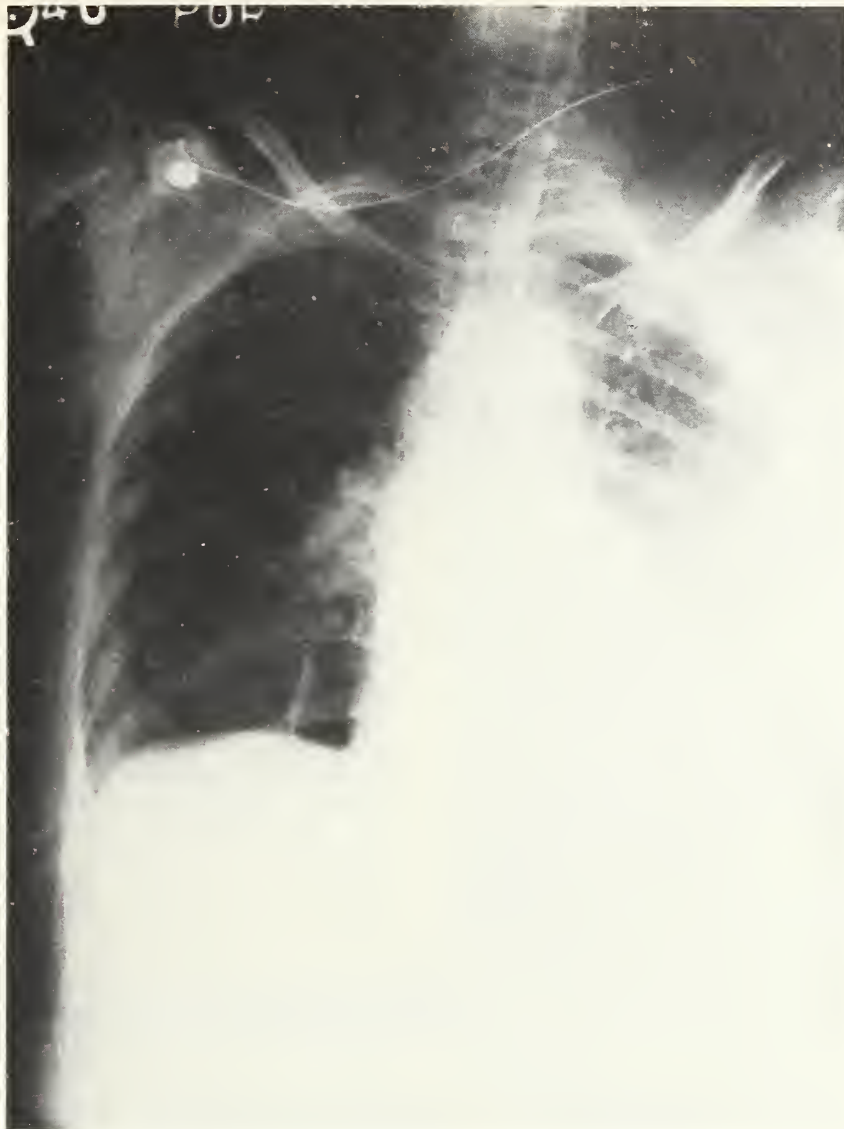


Figure 2

CASE

Figure 1 is a portable AP 40 inch chest film of a 52 year old female involved in a severe motor vehicle accident (MVA). Study the film carefully. What abnormality do you immediately suspect? Is this entity very common? Is the film suspicious or diagnostic of it? Are any other tests or procedures needed? If so, what? If not, why not?

Ignore the ECG leads, but note the well positioned endotracheal tube at the D5 level. Also, there is some rotation of the patient to the left. The obvious finding is the prominent density in the right lower chest, **simulating** an elevated hemidiaphragm. Did you notice the air density along the medial and lateral margins of this density? Also note that no

definite diaphragm shadow (as seen above a pneumoperitoneum) is evident. No rib fractures or pleural fluid are evident. On the basis of these findings it was felt that the patient had a rupture of the right hemidiaphragm (RRHD) with secondary herniation of liver into chest, the liver being partly outlined by air as a result of an associated lung tear.

The patient was soon taken to the operating room. She had RRHD with liver herniation. The liver was returned to normal position, the diaphragm repaired, an injured spleen removed, and multiple bleeding intra-abdominal vessels ligated. Figure 2 is a portable AP 40 inch post-operative chest film, showing a nearly normal right chest and some hazy infiltrate in the left lower chest.

DISCUSSION

Diaphragm rupture or tear may be caused by direct penetrating injury or blunt non-penetrating injury. It is most often due to MVA or falling from a height. The incidence is estimated at about 4% in patients with several chest/abdominal injuries. Its presence can be masked by these injuries. Suspect it with a combination of rib and pelvic fractures. If not recognized in the acute stage, it may be detected years later on a routine chest film!

The central and posterior portions of the left hemidiaphragm are most commonly involved (up to 20 times more than the right, which is "protected" by the liver).

Most series report a high incidence of associated injuries, including brain, spine, pelvis, etc. The hernia contents depend on size and position of the rupture and may contain omentum, stomach, small and large bowel, spleen, etc., in various combinations. In RRHD there is liver herniation in less than 10%. The diagnosis of acute RRHD with liver herniation may be difficult as this may be mistaken for an intact elevated diaphragm! (The diagnosis of our case was made much easier by the air about the liver!) With left sided rupture, air containing loops

of bowel may be seen in chest. Isolated RRHD, secondary to blunt abdominal trauma, with no other associated injury, apparently has not yet been reported.

Various contrast studies have been used to facilitate the diagnosis, but these may be time consuming. A more desirable approach is an emergency liver scan using technetium Tc 99m sulfur colloid. This is a rapid, safe, and accurate method of diagnosing RRHD with liver herniation. The scan will show the abnormally elevated position of the liver and also, may demonstrate a ring compression defect, where the liver is firmly wedged through the diaphragm.

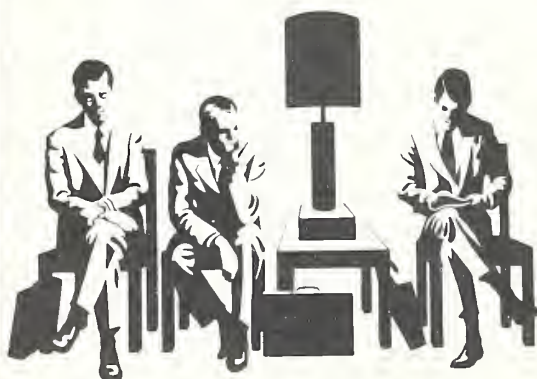
It is common to overlook RRHD in acute injury. It is also not common. Up to 1968, there were less than 40 reported cases. A high index of suspicion will facilitate earlier diagnosis!

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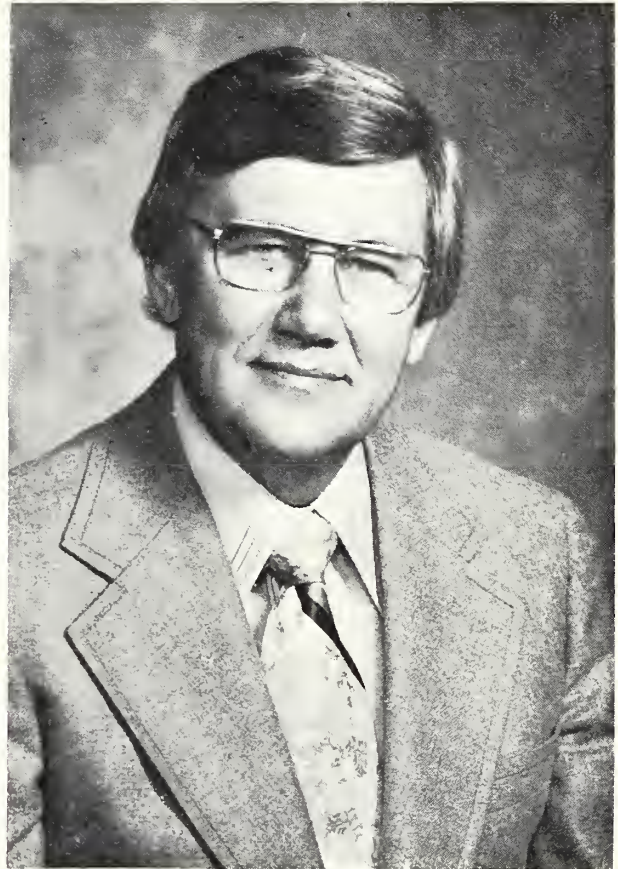
President's Page

As every busy practitioner knows, it is difficult to predict just what medical problems will confront one on a daily or weekly basis, but one thing is predictable: Senator Kennedy will continue to push for National Health Insurance and most likely, President Carter will alter his stand on this same issue, gradually shifting closer to that which Kennedy proposes.

As the Wall Street Journal so nicely described it in a recent editorial, Mr. Kennedy has not quite proposed conscription of doctors but if one would note his bill which would call for a ceiling on total health care spending, the health care industry would finally come under the total control of the federal government.

So we have a plan for economic dictatorship over a selected group of Americans which wins such great approval from other groups as the UAW. Surprisingly, the UAW tells the government to "stay the hell out" of its negotiations with the auto industry this summer. Yet we hear no cries from the UAW, AFL, CIO or even the American Civil Liberties Group when physicians are singled out for complete and total control. From all sides, the liberals continue to espouse the idea that health care is different from other economic endeavors. It is a "right" which the state must guarantee its citizens. They then assume that the only way the state can guarantee this right is through absolute control of the health care industry.

We, as physicians in the state of South Dakota, must continue to warn our clientele that nationalization will not improve medicine in any form. There is great evidence to believe that it will be more costly. Take for example Canada's provincial health system, which Senator Kennedy says is free health care. It has not reduced health care costs to the Canadians. Take the plight of the poor British citizen. Their present system has nearly bankrupted them and has exacted another price from its citizens in the form of long queues for medical care, crum-



bling and outmoded medical facilities and the flight of some of the more able doctors to other hospitable shores. We must make it known that the Kennedy Health Plan would threaten the U. S. with much the same thing. Even more important, is that the human element so crucial to medical practice and so grossly neglected in the technocratic schemes is subverted by political consideration.

Your Association will continue to fight, in its small way, any take over of medicine by the government. We have an obligation to attempt to curtail the ever upward cost of medicine to the patient. Yet, at the same time, it is important that we get the message to our patients that National Health Insurance is a bad way to solve any of our problems that exist in the present system. It simply takes health care in absolutely the wrong direction. It can only lead to mediocrity in the supply of medical services.

Mr. Kennedy's National Health Insurance plan makes us wonder what he has against doctors and to a certain extent, what he has against the sick.

Sincerely

D. B. Reaney, M.D., President
South Dakota State Medical Association



A reminder

ZYLOPRIM[®]

(allopurinol)

100 and 300 mg scored Tablets

- inhibits uric acid formation
- helps prevent urate crystal depositions in synovia
- reduces risk of uric acid lithiasis

INDICATIONS AND USE: This is not an innocuous drug and strict attention should be given to the indications for its use. Pending further investigation, its use in other hyperuricemic states is not indicated at this time.

Zyloprim[®] (allopurinol) is intended for:

1. treatment of gout, either primary, or secondary to the hyperuricemia associated with blood dyscrasias and their therapy;
2. treatment of primary or secondary uric acid nephropathy, with or without accompanying symptoms of gout;
3. treatment of patients with recurrent uric acid stone formation;
4. prophylactic treatment to prevent tissue urate deposition, renal calculi, or uric acid nephropathy in patients with leukemias, lymphomas and malignancies who are receiving cancer chemotherapy with its resultant elevating effect on serum uric acid levels.

CONTRAINDICATIONS: Use in children with the exception of those with hyperuricemia secondary to malignancy. The drug should not be employed in nursing mothers.

Patients who have developed a severe reaction to Zyloprim should not be restarted on the drug.

WARNINGS: ZYLOPRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION. In some instances a skin rash may be followed by more severe hypersensitivity reactions such as exfoliative, urticarial and purpuric lesions as well as Stevens-Johnson syndrome (erythema multiforme) and very rarely a generalized vasculitis which may lead to irreversible hepatotoxicity and death.

A few cases of reversible clinical hepatotoxicity have been noted and in some patients asymptomatic rises in serum alkaline phosphatase or serum transaminase have been observed. Accordingly, periodic liver function tests should be performed during the early stages of therapy, particularly in patients with pre-existing liver disease. Patients should be alerted to the need for due precautions when engaged in activities where alertness is mandatory.

Nevertheless, iron salts should not be given simultaneously with Zyloprim. This drug should not be administered to immediate relatives of patients with idiopathic hemochromatosis.

In patients receiving Purinethol[®] (mercaptopurine) or Imuran[®] (azathioprine), the concomitant administration of 300-600 mg of Zyloprim per day will require a reduction in dose to approximately one-third to one-fourth of the usual dose of mercaptopurine or azathioprine. Subsequent adjustment of doses of Purinethol or Imuran should be made on the basis of therapeutic response and any toxic effects.

Usage in Pregnancy and Women of Childbearing Age. Zyloprim[®] (allopurinol) should be used in pregnant women or women of childbearing age only if the potential benefits to the patient are weighed against the possible risk to the fetus.

PRECAUTIONS: Some investigators have reported an increase in acute attacks of gout during the early stages of allopurinol administration, even when normal or sub-normal serum uric acid levels have been attained.

It has been reported that allopurinol prolongs the half-life of the anticoagulant, dicumarol. This interaction should be kept in mind when allopurinol is given to patients already on anticoagulant therapy, and the coagulation time should be reassessed.

A fluid intake sufficient to yield a daily urinary output of at least 2 liters and the maintenance of a neutral or, preferably, slightly alkaline urine are desirable to (1) avoid the theoretic possibility of formation of xanthine calculi under the influence of Zyloprim therapy and (2) help prevent renal precipitation of urates in patients receiving concomitant uricosuric agents.

Patients with impaired renal function require less drug and should be carefully observed during the early stages of Zyloprim administration and the drug withdrawn if increased abnormalities in renal function appear.

In patients with severely impaired renal function, or decreased urate clearance, the half-life of oxipurinol in the plasma is greatly prolonged. Therefore, a dose of 100 mg per day or 300 mg twice a week, or perhaps less, may be sufficient to maintain adequate xanthine oxidase inhibition to reduce serum urate levels. Such patients should be treated with the lowest effective dose, in order to minimize side effects.

Mild reticulocytosis has appeared in some patients.

As with all new agents, periodic determination of liver and kidney function and complete blood counts should be performed especially during the first few months of therapy.

ADVERSE REACTIONS:

Dermatologic: Because in some instances skin rash has been followed by severe hypersensitivity reactions, it is recommended that therapy be discontinued at the first sign of rash or other adverse reaction (see WARNINGS). Skin rash, usually maculopapular, is the adverse reaction most commonly reported.

Exfoliative, urticarial and purpuric lesions, Stevens-Johnson syndrome (erythema multiforme) and toxic epidermal necrolysis have also been reported.

A few cases of alopecia with and without accompanying dermatitis have been reported.

In some patients with a rash, restarting Zyloprim (allopurinol) therapy at lower doses has been accomplished without untoward incident.

Gastrointestinal: Nausea, vomiting, diarrhea, and intermittent abdominal pain have been reported.

Vascular: There have been rare instances of a generalized hypersensitivity vasculitis or necrotizing angiitis which have led to irreversible hepatotoxicity and death.

Hematopoietic: Agranulocytosis, anemia, aplastic anemia, bone marrow depression, leukopenia, pancytopenia and thrombocytopenia have been reported in patients, most of whom received concomitant drugs with potential for causing these reactions. Zyloprim[®] (allopurinol) has been neither implicated nor excluded as a cause of these reactions.

Neurologic: There have been a few reports of peripheral neuritis occurring while patients were taking Zyloprim. Drowsiness has also been reported in a few patients.

Ophthalmic: There have been a few reports of cataracts found in patients receiving Zyloprim. It is not known if the cataracts predated the Zyloprim therapy. "Toxic" cataracts were reported in one patient who also received an anti-inflammatory agent; again, the time of onset is unknown. In a group of patients followed by Gutman and Yü for up to five years on Zyloprim therapy, no evidence of ophthalmologic effect attributable to Zyloprim was reported.

Drug Idiosyncrasy: Symptoms suggestive of drug idiosyncrasy have been reported in a few patients. This was characterized by fever, chills, leukopenia or leukocytosis, eosinophilia, arthralgias, skin rash, pruritus, nausea and vomiting.

OVERDOSAGE: Massive overdosing, or acute poisoning, by Zyloprim has not been reported.

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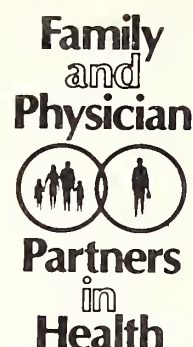
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The attending physician plays an important part in postmortem diagnosis of medico-legal deaths by observing a cardinal rule in pathology—proper specimen handling. Victims of violent or sudden death dying in the hospital environment acquire hospital artifacts which must be delineated to establish the cause and manner of death.

Clothing should remain on or with the body unless given to an investigating officer. Trace evidence recovered may be of little value unless proper chain of custody is preserved. Any disposable items used in resuscitation (IV's, airways, tubing, etc.) should remain in place. Avoid postmortem cleansing of the body. With a detailed record of therapy these precautions should enable separation of resuscitative from pre-hospitalization trauma (ie. IV narcotism). The medications given must be known in order to facilitate post-mortem toxicology. In addition, the documentation of this information serves to protect the physician should future questions concerning therapy be raised. Alteration and probing of wounds, unless medically necessary, should be avoided. A photograph or description of the undisturbed wound is more useful than distorted fragments of tissue in formalin.

With any death having medico-legal implications the body should not be released for embalming without notification of proper legal authorities. This applies whether the individual is dead-on-arrival or dies after prolonged hospitalization. The embalming process restricts toxicologic and microbiologic studies. It also changes the appearance of injuries making it difficult to ever determine the sequence of injury, a matter which may be of crucial import.

An attempt to anticipate and answer questions which may arise in future criminal and civil proceedings should guide the medical aspects of death investigation.

Thomas E. Henry, M.D.
Forensic Pathologist

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Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence:* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy:* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children:* Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdose. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular:* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System:* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal:* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic:* Urticaria, rash, ecchymosis, erythema. *Endocrine:* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System:* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous:* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily one hour before meals, and in mid-evening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSEAGE: Manifestations of acute overdose include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdose.

Product Information as of April, 1976

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References: 1. Citations available on request—Medical Research Department, MERRELL RESEARCH CENTER, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon, R.H., and Leyland, H.M.: A Comprehensive Review of Diethylpropion Hydrochloride. International Symposium on Central Mechanisms of Anorectic Drugs, Florence, Italy, Jan. 20-21, 1977.

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Second Annual Cancer Symposium for West River Area, School of Nursing, 1011 11th St., Rapid City, SD, July 27-28. Fee: \$25. Category I AMA credits. Contact: A. A. Lampert, M.D., 1018 11th St., Rapid City, SD 57701 (605) 394-3064.

Coronary Disease, Exercise Testing, and Cardiac Rehabilitation, Playboy Resort, Lake Geneva, WI, July 27-29. Fee: \$202. 13 hrs. AMA Category I credits. Contact: International Med., Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

August

Black Hills Summer Seminar, Holiday Inn of the Northern Hills, Spearfish, SD, Aug. 9-11. AMA and AAFP credits. Contact: Black Hills Summer Seminar, c/o SDSMA, 608 West Ave., N., Sioux Falls, SD 57104.

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, Four Seasons, Lake of the Ozarks, MO, Aug. 10-12. 13 hrs. AMA Category I credits. Fee: \$215. Contact: Internat'l. Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Medical Staff Law and Bylaws, The Abbey on Lake Geneva, Lake Geneva, WI, Aug. 13-15. Fee: \$350. Contact: Registrar, Aspen Systems Corp., 20010 Century Blvd., Germantown, MD 20767

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, Inn on the Park, Toronto, Canada, Aug. 17-19. Fee: \$215. 13 hrs. AMA Category I credits. Contact: Internat'l Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Mackinac Island Conference, The Grand Hotel, Mackinac Island, MI, Aug. 24-27. AMA Category I credits. Contact: Office of CE, Towsley Center for CME, U. of Michigan Med. School, Ann Arbor, MI 48109.

Medical Office Management Institute Workshops, Radisson South Hotel, Minneapolis, MN, Aug. 27-30. Fee:

\$350. Contact: Conomikes Associates, Inc., 4270 Promenade Way, Suite 122, Marina del Ray, CA 90291, toll free (800) 421-6521.

September

American Cancer Society, National Conference—Breast Cancer 1979, Waldorf-Astoria Hotel, New York, NY, Sept. 6-8. 16 hrs. AMA Category I credits. Contact: Breast Cancer Conf., Am. Cancer Soc., 777 Third Ave., New York, NY 10017.

AMA CME/Golf Program, Pinehurst Hotel and Country Club, Pinehurst, NC, Sept. 16-22. AMA Category 1 credits. Contact: Dept. of Meeting Management, AMA, 535 N. Dearborn St., Chicago, IL 60610.

Mayo Foundation Outreach Seminar, McKennan Hosp. Aud., Sioux Falls, SD, Sept. 28-29. AMA Category 1 credits and AAFP credits. Contact: Off. of Med. Ed., McKennan Hosp., 800 E. 21st St., Sioux Falls, SD 57101.

Coronary Disease, Exercise Testing and Cardiac Rehabilitation, Sheraton on the Wharf, San Francisco, CA, Sept. 28-30. Fee: \$202. 13 hrs. AMA Category I credits. Contact: Internat'l Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

Cardiac Symptoms, Arrhythmias, and Holter Monitoring, Sahara Hotel, Las Vegas, NV, Sept. 28-30. Fee: \$215. 13 hrs. AMA Category I credits. Contact: Internat'l Med. Ed. Corp., 64 Inverness Dr., E., Englewood, CO 80112.

October

American Society of Clinical Pathologists, College of American Pathologists, Las Vegas, NV, Oct. 25-Nov. 2. Contact: ASCP, 2100 W. Harrison St., Chicago, IL 60612.

Mayo Foundation Outreach Seminar, McKennan Hosp. Aud., Sioux Falls, SD, Oct. 26-27. AMA Category 1 credits and AAFP credits. Contact: Off. of Med. Ed., McKennan Hosp., 800 E. 21st St., Sioux Falls, SD 57101.

Omaha Mid-West Clinical Society 47th Annual Postgraduate Assembly, Omaha Hilton Hotel, Omaha, NE, Oct. 29-31. Category I credits. Contact: Lorraine E. Seibel, Ex. Sec., Omaha Mid-West Clinical Society, 7363 Pacific St., Suite 210A, Omaha, NE 68114.

November

45th Annual Scientific Assembly of the American College of Chest Physicians, Hyatt Regency Hotel and the Albert Thomas Convention Hall, Houston, Tex., Nov. 4-8. 30 hrs. AMA Category 1 credits. Contact: Dale E. Braddy, Dir. of Ed., ACCP, 911 Busse Highway, Park Ridge, IL 60068.

Symposium: The Biology and Management of Surgical Wounds, the New York Hosp.-Cornell Med. Center, New York, NY, Nov. 8-9. Contact: Peter Dineen, M.D., Dept. of Surgery, the New York Hosp.-Cornell Med. Center, 1300 York Ave., New York, NY 10021.

Status of Curability of Childhood Cancers, Shamrock Hilton Hotel, Houston, Tex., Nov. 8-10. Contact: Stephen C. Stuyck, Inf. Coord., M.D. Anderson Hosp. and Tumor Instit., Houston, Tex. 77030, (713) 792-3030.

Patient Safety/Risk Control, The Breakers, Palm Beach, FL, Nov. 9-10. Fee: \$200 for AMA members; \$300 for nonmembers, 14 hrs. AMA Category I credits. Contact: Dept. of Hospitals & Health Facilities, AMA, 535 North Dearborn St., Chicago, IL 60610.

Mayo Foundation Outreach Seminar, McKennan Hosp. Aud., Sioux Falls, SD, Nov. 16-17. AMA Category 1 credits and AAFP credits. Contact: Off. of Med. Ed., McKennan Hosp., 800 E. 21st St., Sioux Falls, SD 57101.

December

Fourth Southeastern Conference on Alcohol and Drug Abuse, Downtown Marriott Hotel, Atlanta, GA, Dec. 5-9. 27 hrs. AMA Category I credits. Contact: Conway Hunter, M.D., Fourth SECAD, Peachford Hosp., 2151 Peachford Rd., N.E., Atlanta, GA 30338.

SOUTH DAKOTA JOURNAL OF MEDICINE

Published Monthly by the S.D. State Medical Assn.

Volume XXXII August 1979 Number 8



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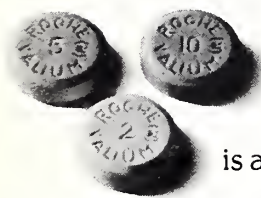
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Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. **Adults:** Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. **Geriatric or debilitated patients:** 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) **Children:** 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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FEATURES

- 7** Transactions of the South Dakota
State Medical Association,
Ninety-Eighth Annual Meeting
- 27** President's Page
- 29** South Dakota AFP Chapter News
- 37** Laboratory Aids
- 43** This Is Your Medical Association
- 44** Editorial
- 47** Roster
- 54** Future Meetings

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Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma, agitated states. Patients with a history of drug abuse. Ouring or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

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PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular:* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System:* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal:* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic:* Urticaria, rash, ecchymosis, erythema. *Endocrine:* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System:* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous:* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

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Product Information as of April, 1976

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Frank Messner, M.D. (1980)Yankton

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Roger Millea, M.D. (1981)Rapid City

Robert L. Ferrell, (1982)Rapid City

A. J. Barrett, M.D. (1980)Rapid City

Tenth District (Rosebud)

M. George Thompson, D.O. (1982)Gregory

Eleventh District (Northwest)

James Wunder, M.D. (1982)Mobridge

Twelfth District (Whetstone Valley)

Eldon Bell, M.D. (1982)Webster

Student Representative

Stewart AbbotYankton

ALTERNATE COUNCILORS

First District (Aberdeen)

G. H. Steele, M.D. (1980)Aberdeen

Second District (Watertown)

James Larson, M.D. (1980)Watertown

Third District (Brookings-Madison)

A. A. Lampert, Jr., M.D. (1981)Madison

Fourth District (Pierre)

M. R. Cosand, M.D. (1980)Pierre

Fifth District (Huron)

G. Robert Bell, M.D. (1981)DeSmet

Sixth District (Mitchell)

C. D. Monson, M.D. (1981)Parkston

Seventh District (Sioux Falls)

Michael Pekas, M.D. (1981)Sioux Falls

J. S. Devick, M.D. (1980)Colton

Richard Tschetter, M.D. (1980)Sioux Falls

D. G. Ortmeier, M.D. (1982)Sioux Falls

Eighth District (Yankton)

Thomas Johnson, M.D. (1982)Yankton

(1980)

Ninth District (Rapid City)

N. R. Whitney, M.D. (1982)Rapid City

Bruce Allen, M.D. (1980)Rapid City

(1981)

Tenth District (Rosebud)

John Malm, M.D. (1982)Gregory

Eleventh District (Northwest)

L. M. Linde, M.D. (1982)Mobridge

Twelfth District (Whetstone Valley)

Joseph Kass, M.D. (1982)Rosholt

Student Representative

1979-1980 COMMISSIONS

COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS

Stephen Haas, M.D., Chairman (1980) Rapid City

V. Janavs, M.D., (1981) Milbank

Ronald Tesch, M.D. (1981) Brookings

John Elston, M.D. (1981) Rapid City

R. W. Honke, M.D. (1981) Wagner

Robert McGee, M.D. (1981) Aberdeen
 C. L. Swanson, M.D. (1980) Pierre
 Bill Church, M.D. (1980) Sioux Falls
 L. W. Karlen, M.D. (1980) DeSmet
 Patrick McGreevy, M.D. (1980) Sioux Falls
 R. B. Henry, M.D. (1982) Brookings
 R. I. Porter, M.D. (1982) Yankton
 J. B. Gregg, M.D. (1982) Sioux Falls
 Walter Baas, M.D. (1982) Mitchell
 Egon Dzintars, Student Member

COMMISSION ON INTERNAL AFFAIRS, COMMUNICATIONS AND LIAISON

Lawrence Finney, M.D., Chairman (1981) Sioux Falls
 Richard Tschetter, M.D. (1981) Sioux Falls
 C. B. Gwinn, M.D. (1981) Rapid City
 Karl Kosse, M.D. (1981) Aberdeen
 William Quick, M.D. (1981) Yankton
 C. R. Stoltz, M.D. (1980) Watertown
 Jeffrey Peterson, M.D. (1980) Mobridge
 L. F. Nelson, M.D. (1980) Webster
 Werner Klar, M.D. (1980) Flandreau
 R. E. Van Demark, M.D. (1980) Sioux Falls
 T. A. Hohm, M.D. (1982) Huron
 Harold Fletcher, M.D. (1982) Vermillion
 Jay Hubner, M.D. (1982) Yankton
 J. F. Barlow, M.D. (1982) Sioux Falls
 Allan Hartzell, M.D. (1982) Sioux Falls
 Patti Vanderlaan, Student Member

COMMISSION ON MEDICAL SERVICE

Howard Saylor, M.D., Chairman (1981) Huron
 Charles Hollerman, M.D. (1981) Yankton
 Warren Jones, M.D. (1981) Sioux Falls
 Guy Tam, M.D. (1981) Sioux Falls
 Larry Ebbert, M.D. (1981) Rapid City
 Lowell Swisher, M.D. (1980) Kadoka
 J. A. Rud, M.D. (1980) Watertown
 Roscoe Dean, M.D. (1980) Wessington Springs
 J. A. Eckrich, Jr., M.D. (1980) Aberdeen
 Anthony Javurek, M.D. (1980) Deadwood
 Kennon Broadhurst, M.D. (1982) Aberdeen
 Curtis Wait, M.D. (1982) Brookings
 Anton Petres, M.D. (1982) Salem
 C. D. Monson, M.D. (1982) Parkston
 John Hoskins, M.D. (1982) Sioux Falls
 Barry Bauer, Student Member

COMMISSION ON SCIENTIFIC MEDICINE

J. C. Larson, M.D., Chairman (1982) Watertown
 Loren Amundson, M.D. (1981) Sioux Falls
 G. Robert Bell, M.D. (1981) DeSmet
 R. J. Foley, M.D. (1981) Tyndall
 Joseph Kass, M.D. (1981) Rosholt
 R. B. Leander, M.D. (1981) Sioux Falls
 Juan Chavier, M.D. (1980) Aberdeen
 T. A. Angelos, M.D. (1980) Canton
 R. R. Thornton, M.D. (1980) Yankton
 Robert Ferrell, M.D. (1980) Rapid City
 R. D. Bloemendaal, M.D. (1980) Rapid City
 Larry Sittner, M.D. (1982) Sioux Falls
 A. J. Janusz, M.D. (1982) Aberdeen
 B. T. Otey, M.D. (1982) Flandreau
 Gene Koob, M.D. (1982) Sioux Falls
 Marsha Beckman, Student Member

PROFESSIONAL LIABILITY COMMISSION

Michael Rost, M.D., Chairman (1980) Sioux Falls
 Dale Berkebile, M.D. (1981) Rapid City

E. W. Sanderson, M.D. (1981) Sioux Falls
 Morris Radack, M.D. (1981) Yankton
 James Hovland, M.D. (1980) Aberdeen
 Donald Kelley, M.D. (1980) Rapid City
 Frank Alvine, M.D. (1982) Sioux Falls
 A. A. Lampert, Jr., M.D. (1982) Madison
 Richard Wake, M.D. (1982) Brookings

CREDENTIALS COMMISSION AND EXECUTIVE COMMISSION

Duane Reaney, M.D., Yankton
 Winston Odland, M.D., Aberdeen
 Bruce Lushbough, M.D., Brookings
 Joseph Hamm, M.D., Sturgis
 Durward Lang, M.D., Sioux Falls
 Russell Harris, M.D., Rapid City
 Richard Gere, M.D., Mitchell

GRIEVANCE COMMISSION

G. E. Tracy, M.D., Chairman (1981) Watertown
 R. E. Van Demark, M.D. (1980) Sioux Falls
 Fred Leigh, M.D. (1982) Huron
 James Ryan, M.D. (1983) Mobridge
 Russell Harris, M.D. (1984) Rapid City

LONG RANGE PLANNING COMMITTEE

T. H. Sattler, M.D., Chairman, Yankton
 Karl Wegner, M.D., Vermillion
 C. E. Tesar, M.D., Rapid City
 Dennis Johnson, M.D., Sioux Falls
 H. J. Stensrud, M.D., Madison
 Michael Pekas, M.D., Sioux Falls
 W. Nicol Guddal, M.D., Watertown
 Jay Bachmayer, M.D., Aberdeen

REPORT OF THE BUDGET AND AUDIT COMMITTEE

12:30 p.m.

Thursday, June 7, 1979

Howard Johnson Motor Lodge
 Rapid City, South Dakota

The meeting was called to order by Larry Finney, M.D., Chairman of the Budget and Audit Committee. Present for roll call were Drs. Finney, Russell Harris, Winston Odland, Duane Reaney, Joseph Hamm, Bruce Lushbough and James Ryan. Also in attendance were Robert Johnson and Patty Butler.

The Committee reviewed the CPA Audit prepared by McGladrey and Hendrickson of Sioux Falls. The Committee recommended that the audit continue to be prepared on a cash basis. A discussion was held on the accumulated equity and a question was raised as to whether or not the interest income could affect the tax status of the Association. The Executive Secretary was directed to obtain information on the cost of the lot to the north of the Association Building and also to obtain the price of other real estate for possible consideration to purchase for the Association. Dr Odland moved that the Committee recommend to the Council that the CPA audit be accepted and approved. The motion was seconded and carried.

Mr. Johnson discussed the financial situation of the WPPA and the problems in the Wyoming PSRO organization. The Committee directed that Mr. Johnson confer with the Association's counsel, Mr. David Gerdes, on the position and future action of the SDSMA in regard to this situation. Dr. Hamm moved that Mr. Johnson report to the Council on the problem.

A discussion was held on the in-state travel allowance provided to the president of the South Dakota State Medical Association. Dr. Lushbough moved that the Committee recommend that the allowance be increased from \$500 per year to \$1000 per year, beginning with the present fiscal year. The motion was seconded and carried.

The Committee reviewed the request for endorsement of the Rural Health Initiative project in Estelline, S.D. Dr. Hamm moved that the proposal be forwarded to the Watertown District Medical Society for review and recommendation, in addition to the information received from the Brookings-Madison District Medical Society, before action is taken by the SDSMA. The motion was seconded and carried.

The meeting adjourned at 2:30 p.m.

FIRST COUNCIL MEETING MINUTES

2:30 p.m. **Howard Johnson Motor Lodge**
Thursday, June 7, 1979 **Rapid City, South Dakota**

The meeting was called to order by Bruce Lushbough, M.D., Chairman of the Council. Those present for roll call were Doctors Russell Harris, Duane Reaney, Winston Odland, Joseph Hamm, W. R. Taylor, Gerald Tracy, Bruce Lushbough, Durward Lang, James Ryan, G. Robert Bartron, R. C. Jahraus, David Buchanan, Richard Gere, W. O. Rossing, Paul Aspaas, D. G. Ortmeier, Gordon Held, Frank Messner, N. R. Whitney, Robert Ferrell, A. J. Barrett, James Wunder, Eldon Bell, student councilor Stewart Abbot, and commission chairmen Lawrence Finney, Howard Saylor, J. C. Larson and T. H. Sattler. Also in attendance were A.A. Lampert, M.D., Charles Hollerman, M.D. and Mr. Don Brekke.

Dr. Aspaas moved to accept the minutes of the previous meeting as printed and distributed. The motion was seconded and carried.

Dr. Lushbough recognized Dr. D. G. Ortmeier serving as alternate councilor for the Seventh District and student councilor, Stewart Abbot.

I. PROGRESS REPORT ON MATERNAL AND CHILD HEALTH PROJECT IN RAPID CITY. Dr. A. A. Lampert presented this report on behalf of the Ad Hoc Committee appointed to study this project. He indicated that a final report would be forthcoming in the near future and would be submitted to the State Medical Association for dissemination or whatever action the Council determines. The members of the committee include Drs. Lampert, Robert Ferrell, A. J. Barrett, G. E. Tracy and N. R. Whitney.

Pros

1. Statistics indicate a need for such a program for the non-white population.
2. Tests available at the MCH Center are available on prescription to patients of other private practice physicians without the patients becoming a part of the MCH program.

Cons

1. Program is available to all who meet criteria regardless of income.
2. There is no advisory committee of practicing physicians in the area as mandated by federal regulation.
3. Center is billing patients even though regulations say no charges will be made.
4. Cost per patient is several times the cost for patients seeing private practice physicians.

This was submitted to the Council for information only.

II. REPORT ON MEDICAL SCHOOL. Dean Hollerman presented information on the NCA visit for the USD School of Medicine. He also outlined the proposed objectives for the School of Medicine for 1980. Dr. Harris encouraged the councilors to talk to local physicians to promote a united front for the continuation of the School of Medicine with its goal to graduate family practice orientated physicians, and to then project this to local legislators.

III. AHEC. Don Brekke presented a program on the AHEC project. He reviewed the premises and objectives of the AHEC and encouraged physicians to become knowledgeable and involved in the implementation of this project in South Dakota. This was submitted to the Council for their information.

IV. WESTERN PHYSICIAN PURCHASING ASSOCIATION. Mr. Johnson reported that the WPPA is having difficulty in membership recruitment and that he will give the Council an update following the next Board of Directors meeting.

V. REVIEW OF ANNUAL MEETING SCHEDULE. Mr. Johnson reviewed the annual meeting schedule for the councilors' information.

VI. LETTER FROM INTERAGENCY COUNCIL ON SMOKING. The Interagency Council requested the State Medical Association's endorsement for a petition drive to gather enough signatures to obtain a referendum vote in the 1980 election to ban smoking in certain designated places.

Pros

1. Physicians are concerned that smoking is harmful.

Cons

1. Concern that this discriminates against peoples' habits and life styles.
2. Regulations of this type are the prerogative of the Legislature.

Dr. Bell moved that the State Medical Association endorse the petition of the South Dakota Interagency Council on Smoking and Health to obtain a referendum vote in the 1980 election to ban smoking in certain designated places. The motion was seconded and failed.

VII. EMERGENCY MEDICAL SERVICE. Dr. Saylor reported that the Division of Emergency Medical Services, Department of Health, is submitting a grant application for an advanced life support system in northeast South Dakota. He stated that the grant application will be submitted to the physicians for review and recommendation before it is submitted to the federal government for approval and funding. He encouraged the Liaison Committee to the Health Department to consider this in their upcoming deliberations with representatives of the Health Department inasmuch as the Division anticipates submitting the grant proposal by October 30, 1979. This was submitted to the Council for information only.

VIII. USD MEDICAL SCHOOL AND RESIDENCY PROGRAMS. Dr. Bartron addressed the Council and encouraged the physicians to develop and project a united front regarding the continuation of the Medical School and the residency programs in South Dakota. It was noted that the Yankton District Medical Society was introducing a resolution to the House of Delegates which would address this and hopefully project the consensus of the physicians in South Dakota.

IX. REPORT OF THE BUDGET AND AUDIT COMMITTEE. The Budget and Audit Committee recommended that the report of the Association's auditing firm be accepted by the Council. **Dr. Ortmeier moved that the Council accept the report of McGladrey Hendrickson & Company, the Association's auditing firm. The motion was seconded and carried.**

The Budget and Audit Committee recommended that the State Association President's in-state travel allowance be increased from \$500 to \$1,000 annually. **Dr. Taylor moved that the Council increase the President's in-state travel allowance to \$1,000 annually. The motion was seconded and carried.**

X. RESOLUTION CONCERNING INFANT FORMULA ACTION COALITION "INFAC" SUBMITTED BY H. BEN MUNSON, M.D. **Dr. Ferrell moved that this resolution be referred to the appropriate commission for review and recommendation. The motion was seconded and carried.**
The meeting adjourned at 5:00 p.m.

SECOND COUNCIL MEETING MINUTES

10:45 a.m. Howard Johnson Motor Lodge
Sunday, June 10, 1979 Rapid City, South Dakota

The meeting was called to order by Bruce Lushbough, M.D., chairman.

Those present for roll call included Doctors Duane Reaney, Winston Bryant Odland, Bruce Lushbough, Durward Lang, W. R. Taylor, G. E. Tracy, Russell Harris, Joseph Hamm, G. Robert Bartron, R. C. Jahraus, David Buchanan, Richard Gere, W. O. Rossing, Paul Aspaas, John F. Barlow, Gordon Held, Frank Messner, Roger Millea, A. J. Barrett, Robert Ferrell, M. George Thompson, Eldon Bell, James Wunder, and commission chairmen Doctors Howard Sayl and J. C. Larson and student representative Stewart Abbot.

Dr. Buchanan moved to dispense with the reading of the minutes of the previous meeting inasmuch as they will be published and distributed.

Dr. Lushbough welcomed Dr. M. George Thompson, the new councilor from the Rosebud District, and Stewart Abbot, the new student councilor, and Randy Hart, guest. He also introduced Jim Kosmo from the AMA office.

I. ELECTION OF CHAIRMAN OF THE COUNCIL. Dr. Messner nominated Dr. R. G. Gere. The nomination was seconded. Dr. Jahraus nominated Dr. David Buchanan. The nomination was seconded. There being no further nominations, a ballot was cast. **Dr. R. G. Gere was declared chairman of the council** and he assumed that position immediately.

II. SCHEDULE OF COUNCIL MEETINGS FOR 1979-1980. **Dr. Bell moved to accept the meeting schedule as proposed.** The motion was seconded and carried.

III. ELECTION OF SECRETARY-TREASURER. Dr. Harris nominated Dr. Joseph Hamm. The nomination was seconded. There being no further nominations. **Dr. Hamm was declared secretary-treasurer for a three year term.**

The meeting adjourned at 11:00 a.m.

REMARKS BY JOHN J. COURY, JR., M.D.

Immediately following the Council meeting, John J. Coury, Jr., M.D., a member of the AMA Board of Trustees, addressed members of the State Medical Association. He discussed matters in which the AMA is involved including national legislation, cost containment activities and what can be done individually at a local level, continuing medical education programs, and legal issues such as FTC rulings and chiropractice lawsuits where the AMA is representing organized medicine. He also reviewed the AMA's financial situation briefly and indicated that in the near future dues will have to be increased if physician membership is not increased substantially. He stressed the importance of involving students and residents in local, state and AMA activities inasmuch as they are medicine's future and should be involved in decisions concerning the many issues facing medicine today. Dr. Coury encouraged physicians to share their opinions and ideas with the AMA

officers and Board members so that matters of importance on a local level can be understood and dealt with appropriately at the national level. Dr. Odland stated that the South Dakota State Medical Association appreciates the personal contacts by AMA officers and Board members such as Dr. Coury's visit and feels this is perhaps the best way to keep in touch with physicians throughout the country at a local level.

MINUTES OF THE FIRST HOUSE OF DELEGATES MEETING

8:45 a.m. Howard Johnson Motor Lodge
Friday, June 8, 1979 Rapid City, South Dakota

The meeting was called to order at 8:45 a.m. by Durward Lang, M.D., Speaker of the House. Mr. Robert Johnson, Executive Secretary, called the roll. The following officers, councilors, delegates and alternate delegates were present: Doctors Russell Harris, Duane Reaney, Winston Odland, Joseph Hamm, Durward Lang, William R. Taylor, Gerald E. Tracy, James Ryan, G. Robert Bartron, Bruce Lushbough, R. C. Jahraus, David Buchanan, R. G. Gere, Paul Aspaas, Denny Ortmeier, William Rossing, Frank Messner, Gordon Held, A. J. Barrett, N. R. Whitney, James F. Wunder, Eldon Bell, Paul Leon, A. J. Janusz, James Rud, James Larson, J. A. Muggly, Werner Klar, L. C. Askwig, T. A. Hohm, Charles Monson, Walter Baas, Guy Tam, Bill Church, L. J. Hyland, D. L. Johnson, R. O. Wyatt, L. W. Finney, Gail Benson, William Dendinger, Robert Neumayr, Lee Ahrlin, C. E. Tesar, Thomas Mead, William Jones, Thomas Henry, M. George Thompson, David Yecha, E. A. Johnson, and student representatives Stewart Abbot, Tim Zoellner, and Randy Hart.

Dr. Harris, president of the South Dakota State Medical Association addressed the House of Delegates on matters of concern and interest to the State Medical Association.

Dr. Harris presented the following awards:

50 year awards: G. J. Bloemendaal, M.D. and A. P. Peeke, M.D.

Special Presidential Award—G. Robert Bartron, M.D.

C. B. Alford Award—J. D. Bailey, M.D.

AMPAC Award—to SoDaPAC for being the top PAC in the country in the category of contribution per member.

AMA-ERF Grant—presented by Mrs. J. P. Steele to Charles Hollerman, M.D.

Past President's Award—James Ryan, M.D.

Community Service Award—C. J. McDonald, M.D.

Distinguished Service Award—Helen Jane Hare, M.D.

Army Meritorious Service Medal—presented to Eldon Bell, M.D. by Major General Duane Corning.

Dr. Tracy moved to dispense with the reading of the minutes of the previous meeting inasmuch as they have been published in the South Dakota Journal of Medicine. The motion was seconded and carried.

Dr. Lang appointed G. Robert Bartron, M.D. to act as parliamentarian for the House of Delegates.

Dr. Lang announced the appointments to the Nominating Committee to be as follows:

District 1—A. J. Janusz, M.D.

District 2—James Rud, M.D.

District 3—Werner Klar, M.D.

District 4—Marion Cosand, M.D.

District 5—David Buchanan, M.D.

District 6—Charles Monson, M.D.

District 7—Dennis Johnson, M.D.

District 8—Frank Messner, M.D.

District 9—A. J. Barrett, M.D., Chairman

District 10—John Malm, M.D.

District 11—James Wunder, M.D.

District 12—Eldon Bell, M.D.

Dr. Lang then announced his appointments to the four reference committees.

Reference Committee on Credentials, Resolutions and Memorials, and Reports of Officers and Councilors

Ronald Wyatt, M.D., Chairman

Lee Ahrlin, M.D.

David Yecha, M.D.

Reference Committee on Reports of Commissions on Medical Service, Legislation and Governmental Relations

T. A. Hohm, M.D., Chairman

G. H. Steele, M.D.

William Dendinger, M.D.

Reference Committee on Reports of Commissions on Scientific Medicine, Internal Affairs, Communications and Liaison

E. A. Johnson, M.D., Chairman

Walter Baas, M.D.

George Thompson, D.O.

Reference Committee on Reports of Special Committees and Miscellaneous Business

Thomas Mead, M.D., Chairman

James Larson, M.D.

Paul Leon, M.D.

Dr. Klar moved to dispense with the reading of the reports of the President, President-Elect, Vice President, Secretary-Treasurer, Delegate and Alternate Delegate to the AMA, Executive Secretary, Speaker of the House, Councilor at Large, Chairman of the Council and Councilors inasmuch as they have been published in the handbook. The motion was seconded and carried.

Dr. Held then introduced a resolution from the Yankton District Medical Society which was accepted as resolution #3 and referred to the Reference Committee on Reports of Special Committees and Miscellaneous Business.

RESOLUTION #3

TO: House of Delegates
South Dakota State Medical Association
FROM: Yankton District Medical Society
SUBJECT: USD School of Medicine
REFERRED TO:

WHEREAS, the University of South Dakota School of Medicine has recently received three year accreditation by the LCME which demonstrates the overall high caliber of the program at the USD School of Medicine, and,

WHEREAS, the recognized primary objective of the South Dakota School of Medicine is a family practice oriented curriculum, and,

WHEREAS, the School of Medicine needs the continued support of the Board of Regents, South Dakota Legislature, and the citizens of South Dakota financially and otherwise, and,

WHEREAS, this continued support is essential to continue the many positive programs of the School, and,

WHEREAS, the residencies presently providing training in our state have complemented, and in fact, played an integral part in the overall success of the USD School of Medicine, and do in fact complement and support the family practice orientation of the School. The positive impact that these residencies and the School will have on the quality and accessibility of care in our state must continue. Now, therefore,

BE IT RESOLVED, that the South Dakota State Medical

Association reaffirms its active support of the USD School of Medicine, and its objective of family practice orientation and the residency programs presently operating; this support to be stated in writing to the Board of Regents, the South Dakota Legislature, the Governor, the general public, and all other interested parties.

Dr. Messner, Councilor from the Yankton District Medical Society, then announced that Resolution #2, submitted by the Yankton District Society, was being withdrawn as a result of action taken by that District.

Dr. Lang referred the reports contained in the Handbook to the appropriate Reference Committees. He read Resolution #1, concerning a change in the Bylaws

RESOLUTION #1

BYLAW REVISION

TO: House of Delegates
South Dakota State Medical Association
FROM: The Council
SUBJECT: Continuing medical education requirement for membership in South Dakota State Medical Association

REFERRED TO:

WHEREAS, 150 continuing medical education hours every three years are now a requirement for membership in the South Dakota State Medical Association; and

WHEREAS, Category I accreditation of South Dakota's twelve regional centers has not proceeded as planned due to problems nationally in the accreditation procedure; and

WHEREAS, there is overlap and confusion in CME requirements for various organizations and specialty societies to which South Dakota physicians belong;

THEREFORE, BE IT RESOLVED that Article III, Membership Section I, requirements beginning at line 7 be amended as follows:

Active members of the Association shall be required to complete 150 hours of defined continuing medical education credits over each three year period time for maintaining membership; such specified hours to be approved by a recognized accrediting body. The Continuing Medical Education Committee of the South Dakota State Medical Association shall have the privilege of waiving this requirement when due cause can be shown by the member. Accumulation of such credit hours shall begin ((January 1, 1977.)) ((January 1, 1979.))

and referred consideration of this matter to the Reference Committee on Reports of Special Committees and Miscellaneous Business. Dr. Lang made several announcements concerning the schedule of events for the annual meeting. A film was shown which has been produced by the Multnomah County Medical Society, Portland, Oregon, on how the public views doctors.

The meeting adjourned at 9:30 a.m.

MINUTES OF THE SECOND HOUSE OF DELEGATES MEETING

9:30 a.m. Howard Johnson Motor Lodge
Sunday, June 10, 1979 Rapid City, South Dakota

The meeting was called to order by Durward Lang, M.D., Speaker of the House.

Present for roll call were Doctors Russell Harris, Duane Reaney, Winston Odland, Joseph Hamm, Durward Lang,

William Taylor, Gerald Tracy, James Ryan, G. Robert Bartron, Bruce Lushbough, R. C. Jahraus, David Buchanan, R. G. Gere, Paul Aspaas, William Rossing, John Barlow, Frank Messner, Gordon Held, A. J. Barrett, Roger Millea, Robert Ferrell, James Wunder, Eldon Bell, Paul Leon, A. J. Janusz, James Rud, James Larson, J. A. Muggly, Werner Klar, L. C. Askwig, G. M. Huet, Theodore Hohm, Walter Baas, Guy Tam, Bill Church, L. J. Hyland, Dennis Johnson, Gail Benson, Robert Neumayr, Lee Ahrlin, C. E. Tesar, David Smith, Joseph Kovarik, Joseph Sejvar, M. George Thompson, David Yecha, E. A. Johnson and students Stewart Abbot, Tim Zoellner and Randy Hart.

Dr. Harris moved to dispense with the reading of the minutes of the previous meeting inasmuch as they will be published. The motion was seconded and carried.

The report of the Nominating Committee was read by Dr. James Rud.

REPORT OF THE NOMINATING COMMITTEE

The Nominating Committee submits the following recommendations for the consideration of the House of Delegates:

COUNCILORS

Sioux Falls District #7

R. E. Gunnarson, M.D. (3 year term)

Yankton District #8

Gordon Held, M.D. (3 year term)

Black Hills District #9

Robert Ferrell, M.D. (3 year term)

Rosebud District #10

George Thompson, D.O. (3 year term)

Northwest District #11

James Wunder, M.D. (3 year term)

Whetstone Valley District #12

Eldon Bell, M.D. (until further notified by the District)

ALTERNATE COUNCILORS

Sioux Falls District #7

D. G. Ortmeier, M.D. (3 year term)

Yankton District #8

Thomas Johnson, M.D. (3 year term)

Black Hills District #9

Nathaniel Whitney, M.D. (3 year term)

Rosebud District #10

John Malm, M.D. (3 year term)

Northwest District #11

L. M. Linde, M.D. (3 year term)

Whetstone Valley District #12

Joseph Kass, M.D. (until further notified by the District)

OFFICERS

President Elect

Winston Bryant Odland, M.D.

Vice President

Bruce Lushbough, M.D.

Speaker of the House

Durward Land, M.D.

ANNUAL MEETING SITE

1980—Aberdeen—May 30—June 1

1981—Sioux Falls

1982—Rapid City

Respectfully submitted
A. J. Barrett, M.D., Chairman
NOMINATING COMMITTEE

Dr. Klar moved to accept that section of the reference committee report pertaining to councilors. The motion was seconded and carried.

Dr. Aspaas moved to accept that section of the reference committee report pertaining to alternate councilors. The motion was seconded and carried.

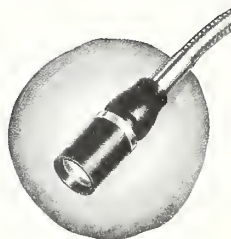
The Speaker called for nominations from the floor for

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SIOUX CITY, IOWA

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Sioux Falls, S.D.
336-1155

219 OMAHA
RAPID CITY, S.D.

the office of president elect. **Dr. Harris moved that nominations cease and a unanimous ballot be cast for Dr. Winston Bryant Odland.** The motion was seconded and carried.

The Speaker called for nominations from the floor for the office of vice president. **Dr. Muggly moved that nominations cease and a unanimous ballot be cast for Dr. Bruce Lushbough.** The motion was seconded and carried.

The Speaker called for nominations from the floor for office of Speaker of the House. **Dr. Rossing moved that nominations cease and a unanimous ballot be cast for Dr. Durward Lang.** The motion was seconded and carried.

Dr. Buchanan moved to accept the sites for upcoming annual meetings as recommended by the reference committee. The motion was seconded and carried.

Dr. Lang introduced Dr. John Coury, a member of the AMA Board of Trustees. Dr. Coury briefly reviewed the AMA budget and dues structure and challenged physicians to promote both state and AMA membership. He stated that the AMA officers and Board members are working hard on behalf of the members, traveling to all areas of the United States to get physicians' feelings, and meeting with corporate opinion makers to promote medicine's views. Dr. Coury invited the House members to attend his briefing which will follow the Second Council meeting.

The report of the Reference Committee on Credentials, Resolutions and Memorials and Reports of Officers and Councilors was read by Dr. Lee Ahrlin.

REPORT OF THE REFERENCE COMMITTEE ON CREDENTIALS, RESOLUTIONS AND MEMORIALS AND REPORTS OF OFFICERS AND COUNCILORS

The following delegates, alternates, officers and councilors of the South Dakota State Medical Association were present: Doctors Russel Harris, Duane Reaney, Winston Odland, Joseph Hamm, Durward Lang, Wm. Taylor, Gerald Tracy, James Ryan, G. Robert Bartron, Bruce Lushbough, R. C. Jahraus, David Buchanan, R. G. Gere, Paul Aspaas, D. G. Ortmeier, Wm. Rossing, Frank Messner, Gordon Held, A. J. Barrett, N. R. Whitney, James Wunder, Eldon Bell, Stewart Abbot, Student Councilor, Paul Leon, A. J. Janusz, James Rud, James Larson, Joseph Muggly, Werner Klar, Charles Monson, Walter Baas, Guy Tam, L. J. Hyland, D. L. Johnson, R. O. Wyatt, L. W. Finney, Wm. Dendinger, Robert Neumayr, Lee Ahrlin, C. E. Tesar, Thomas Mead, Wm. Jones, M. George Thompson, David Yecha, E. A. Johnson, T. Hohm, L. C. Askwig, E. A. Hofer, Gail Benson, Thomas Henry, and Tim Zoellner and Randy Hart, Student Delegates.

A quorum was present for the meeting of the House of Delegates. Total registration for the convention is 286, including 150 physicians, 23 guests, 105 Auxiliary members and 8 sponsors.

The Committee submits the following resolution for the consideration of the House of Delegates:

WHEREAS, the Black Hills District Medical Society, the Black Hills District Auxiliary, the Pierre District Auxiliary and the Mitchell District Auxiliary members have been so thorough in making arrangements for the success of the combined meeting of our 98th anniversary,

BE IT RESOLVED, that the South Dakota Medical Association give its voice in appreciation and thanks to the local physicians in the Black Hills District and the members of the Black Hills District Auxiliary, the Pierre District Auxiliary and the Mitchell District Auxiliary.

WHEREAS, the management of the Howard Johnson Motor Lodge has been so cooperative in providing facilities for the success of the 98th annual meeting of the South Dakota State Medical Association,

BE IT RESOLVED, that the South Dakota State Medical Association extend its thanks and appreciation to the Howard Johnson Motor Lodge.

WHEREAS, the Rapid City Journal, KOTA TV and radio, KTOQ radio and KKLS radio have been most cooperative in presenting the public news of the 98th annual meeting of the South Dakota State Medical Association,

BE IT RESOLVED, that the South Dakota State Medical Association extend its thanks to the Rapid City Journal, KOTA TV and radio, KTOQ radio and KKLS radio.

BE IT RESOLVED, that \$50 be donated to the South Dakota Medical School Endowment Association in memory of the following physicians who died during the past year:

Joseph Lovering, M.D.

Robert A. Buchanan, M.D.

Paul Reagan, M.D.

The Committee reviewed the reports of the officers and councilors and recommends they be accepted as submitted.

The Committee would like to recognize the outstanding work and ability of our Executive Secretary, Robert Johnson and his staff in their continued exemplary performance during the last year in conducting the South Dakota State Medical Association business.

Respectfully submitted,

REFERENCE COMMITTEE ON CREDENTIALS, RESOLUTIONS AND REPORTS OF OFFICERS AND COUNCILORS

Ronald Wyatt, M.D., Chairman

Lee Ahrlin, M.D.

David Yecha, M.D.

Dr. Dennis Johnson moved to accept the report of the Reference Committee on Credentials, Resolutions and Memorials and Reports of Officers and Councilors. The motion was seconded and carried.

The report of the Reference Committee on Reports of the Commission on Medical Service and the Commission on Legislation and Governmental Relations was read by Dr. Theodore Hohm.

REPORT OF THE REFERENCE COMMITTEE ON REPORTS OF THE COMMISSION ON MEDICAL SERVICE AND THE COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS

The Reference Committee carefully reviewed the report of the Commission on Legislation and Governmental Relations. The Reference Committee recommends the acceptance of the report of the Commission on Legislation and Governmental Relations.

The Reference Committee reviewed the report of the Commission on Medical Service. The Reference Committee recommends the acceptance of the report of the Commission on Medical Service.

Respectfully submitted,

REFERENCE COMMITTEE ON REPORTS OF THE COMMISSION ON MEDICAL SERVICE AND THE COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS

Theodore Hohm, M.D., Chairman

G. H. Steele, M.D.

William Dendinger, M.D.

Dr. James Larson moved to accept the report of the Reference Committee on Reports of the Commission on Medical Service and the Commission on Legislation and Governmental Relations. The motion was seconded and carried.

The report of the Reference Committee on Reports of the Commission on Scientific Medicine and the Commission

on Internal Affairs, Communications and Liaison was read by Dr. E. A. Johnson.

REPORT OF THE REFERENCE COMMITTEE ON REPORTS OF THE COMMISSIONS ON SCIENTIFIC MEDICINE AND INTERNAL AFFAIRS COMMUNICATIONS AND LIAISON

The Reference Committee considered the report of the Commission on Scientific Medicine. The Reference Committee recommends the acceptance of the report of the Commission on Scientific Medicine.

The Reference Committee considered the report of the Commission on Internal Affairs, Communications and Liaison. In reviewing the proposed budget for the fiscal year 1979-1980, the Commission took note of the recommendation from the Budget and Audit Committee and the Council that the in-state travel allowance for the president of the South Dakota State Medical Association be increased to \$1,000.00 per year, from \$500.00. This change would increase the item for Physician Travel to \$7,250.00 and decrease the anticipated reserve to \$3,550.00. The Reference Committee recommends that this change be approved. The Reference Committee recommends the acceptance of the report of the Commission on Internal Affairs, Communications and Liaison with this one adjustment.

Respectfully submitted,

REFERENCE COMMITTEE ON REPORTS OF COMMISSIONS ON SCIENTIFIC MEDICINE, AND INTERNAL AFFAIRS, COMMUNICATIONS AND LIAISON

E. A. Johnson, M.D., Chairman

Walter Baas, M.D.

M. George Thompson, D.O.

Dr. Bruce Lushbough moved to accept the report of the Reference Committee on Reports of the Commission on Scientific Medicine and the Commission on Internal Affairs, Communications and Liaison. The motion was seconded and carried.

The report of the Reference Committee on Reports of Special Committees and Miscellaneous Business was read by Dr. Paul Leon.

REPORT OF THE REFERENCE COMMITTEE ON REPORTS OF SPECIAL COMMITTEES AND MISCELLANEOUS BUSINESS

The Reference Committee reviewed the report of the Commission on Professional Liability. The Reference Committee recommends adoption of this report.

The Reference Committee reviewed the report of the Committee for Continuing Medical Education. The Reference Committee recommends adoption of this report.

The Reference Committee reviewed the report of the Grievance Committee. The Reference Committee recommends the adoption of this report and recommends that when the mechanism for the Impaired Physician Program has been established, information on the program be disseminated to the members of the State Medical Association.

The Reference Committee reviewed the report of the Committee on Long Range Planning. The Committee noted for clarification that the AHEC will be supervised by the School of Medicine and encourages physicians in South Dakota to be involved in the implementation of this program. The Committee also recommended the Commission on Scientific Medicine consider including a session on AHEC for the 1980 annual meeting, and that the Councilors be encouraged to bring information on this project to the attention of physicians in their districts. The Reference Committee recommended adoption of this report.

The Reference Committee reviewed the report of the South Dakota Political Action Committee. The Reference Committee recommends adoption of this report.

The Reference Committee reviewed the report of the

Endowment Association. The Reference Committee recommends the adoption of this report.

The Reference Committee reviewed Resolution #1. The Reference Committee recommends the adoption of Resolution #1 and recommends that this matter be referred to the appropriate Commission for study and evaluation.

The Reference Committee reviewed Resolution #3. The Reference Committee recommends the adoption of Resolution #3.

Respectfully submitted,

REFERENCE COMMITTEE ON REPORTS OF SPECIAL COMMITTEES AND MISCELLANEOUS BUSINESS

Thomas Mead, M.D., Chairman

Paul Leon, M.D.

James C. Larson, M.D.

Dr. Bruce Lushbough moved to accept the report of the Reference Committee pertaining to the Report of the Commission on Professional Liability, the Committee for Continuing Medical Education, and the Grievance Commission. The motion was seconded and carried.

Dr. John Barlow moved to accept the report of the Reference Committee pertaining to the Report of the Long Range Planning Committee. The motion was seconded and carried.

Dr. L. J. Hyland moved to accept the report of the Reference Committee pertaining to Reports of the South Dakota Political Action Committee and the South Dakota Medical School Endowment Association. The motion was seconded and carried.

Dr. Bruce Lushbough moved to accept the recommendation of the Reference Committee concerning resolution #1. The motion was seconded and carried.

Dr. Russell Harris moved to accept the recommendation of the Reference Committee concerning resolution #3. The motion was seconded followed by statements indicating the South Dakota Internal Medicine Society and the South Dakota Ophthalmology and Otorhinolaryngology Society had adopted resolutions wholeheartedly supporting the USD School of Medicine. The motion carried.

Dr. Harris thanked the members for the privilege of serving as state president during the past year. He then presented the Presidential Medallion to Dr. Duane Reaney, incoming president. Dr. Lang administered the Oath of Office to Dr. Reaney. Dr. Reaney addressed the House briefly acknowledging the efforts made by Dr. Harris on behalf of organized medicine in South Dakota and pledging his dedication to the ideals and goals of the South Dakota State Medical Association.

Dr. Lang introduced Dr. Winston Bryant Odland, the new president elect, and Dr. Bruce Lushbough, the new vice president. He announced a brief Council meeting would be held immediately following adjournment of the House.

The meeting adjourned at 10:30 a.m.

REPORT OF THE PRESIDENT OF THE SOUTH DAKOTA STATE MEDICAL ASSOCIATION

The past year has been an active one, and I am pleased to say your Medical Association is alive and well. We have many ongoing problems to continue to confront on behalf of the people of our state but they are not insoluble, and with your continued effort and dedication, as you have shown in the past, they can be overcome.

We are extremely fortunate to have the exceptional executive secretary and staff that we have, as all presidents in recent years have attested to. I wish to thank them for their counsel and help through the past year.

It was my pleasant duty to attend both meetings of the AMA House of Delegates in St. Louis and Chicago respectively, accompanying your delegate and alternate delegate, to watch, learn and contribute what I could in representing you. I attended Council meetings, various Com-

mission meetings and presided at Executive Commission meetings. In the fall, your executive secretary and I attended an AMA sponsored meeting on state legislative matters, which had executive secretaries and officers of medical associations from all over the country present. We found that the problems are basically the same in every state in the union; however, some of the larger states especially are having a far more difficult time with theirs.

I also attended the North Central Medical Conference again this year and would, as I have before, urge you to go to these meetings if you can.

The current overview of the national scene and the upper midwest area socio-economic political problems for medicine gives much insight into things to come and progress that may or may not be made from medicine's point of view.

This year's session of the South Dakota State Legislature was overall positive as far as medicine was concerned. A review of the bills of specific interest was presented in the "Grab Bag". The areas that were not as successful as we had hoped developed because of strong pressure for individual rights and freedom to choose, which we considered misguided, but nevertheless prevailed after much debate and amendment.

I have participated in many discussions with the State Health Department concerning mutual interests and specific concerns regarding government and the practice of medicine. The Secretary of Health has expressed strong desire to work with your Medical Association to improve medical care and, in a broader sense, health care for the people of South Dakota. Continued dialogue will be imperative.

Your executive secretary and I met with the Welfare Department and HEW representatives concerning Title XIX provider agreements and, hopefully, the problem is resolved.

We have continued to discuss mutual concerns with the State Hospital Association, and this dialogue should continue.

The progress of national problems such as second opinions, FTC lawsuits against the AMA, hospital rate review and the AMA settlement in a Pennsylvania lawsuit have been monitored to keep you informed as they all have some direct effect on the practice of medicine in South Dakota.

Of continued concern is our relationship with the University of South Dakota School of Medicine and to this end, individual and committee discussions have been ongoing with the Dean and his representatives to try and solve mutual problems. The medical school is turning out a good product that is needed by the people of South Dakota, and I urge each of you to continue your support in all areas.

I want to take this opportunity to thank all of you who have served on the various commissions and committees this past year. It's a lot of work and means much time away from your practices and your families. You served because it is important to all of us and to the people of South Dakota. You have done an outstanding job and I thank you sincerely.

In closing, I would remind you again that the physicians in this country have always been the advocates of the people in medical affairs, and I urge you to continue to be that advocate.

Respectfully submitted,
Russell H. Harris, M.D.
President

The Reference Committee reviewed the report of the President and recommended its approval as submitted.

REPORT OF THE PRESIDENT-ELECT

This office has continued to be a steady learning process in preparation for assumption of the duties as president. Besides attending the usual Executive Committee and

Council meetings, I have had the privilege of attending the AMA Leadership Conference in Chicago in February. Many of the great issues that are facing medicine today, not only in the nation but in South Dakota, were covered in this conference in detail.

It has been a pleasure to serve the State Association in this capacity in the past year.

Respectfully submitted,
D. B. Reaney, M.D.
President-Elect

The Reference Committee reviewed the report of the President-Elect and recommended its approval as submitted.

REPORT OF THE VICE PRESIDENT

During the past year as Vice President of the South Dakota Medical Association, I attended the Council meetings and the Executive Commission meetings and served on the Liaison Committee for the Health Department of the medical school. It has always impressed me of the thoughtful manner in which the proceedings and determinations of the Council have taken place. There is a wide spectrum of business conducted by the Council and Executive Committee and the House of Delegates, all of which is in the ultimate interest of patient care and the preservation of the distinguished quality of the medical profession.

I was frankly encouraged by the manner in which your Medical Association in the state of South Dakota has resisted further invasion by various levels of government and by the well meaning laity. It has previously been said that the true power of the Medical Association can serve only the just interests for a continued effort to improve patient care and cannot stand alone on political or economic issues. The ill-founded concept to produce a mandatory second opinion program across the nation was refuted in this state through the actions of the Executive Committee and the Council. /

Considerable new contact with the medical school has developed during the past year which is beginning to mature the relationship between private physicians and the University of South Dakota School of Medicine; a very mutually beneficial situation for education, patients and private physicians. I have seen the bodies of our Association work sincerely toward this goal. Availability of quality continued medical education has improved greatly in the past several months.

There are indications of expanded roles in the delivery of patient care by the State Health Department. These programs must be carefully evaluated, and those which are not in the long term in the best interests of good patient care and economy for the taxpayer should be rejected. There is no question that the increased involvement of government in medicine and other phases of life in South Dakota constitutes the development of socialism. This should be recognized, and those who feel the need to preserve the Constitution of the United States and the republic it has created should make themselves heard.

The President of the South Dakota Medical Association, Russ Harris, M.D., consistently presented a strong case for the freedom of medicine and also a strong case for developing the highest quality of medical care delivery possible. I would also like to thank the membership, the other officers, members of the Council, the members of the Commissions, the members of special committees and Robert Johnson and his excellent staff for the well spent time and effort on behalf of medicine in South Dakota.

Respectfully submitted,
Winston Bryant Odland, M.D.
Vice President

The Reference Committee reviewed the report of the Vice President and recommended its approval as submitted.

The Maker

Examining a Few Myths About Prescribing.

Increasing pressure is being put on the practicing physician to prescribe drugs generically. You are told that brand-name products are universally "expensive" and generic versions are relatively "cheap." To make this case, the most extreme (rather than typical) price differentials are cited. Thus, consumers are led to believe that such differentials are commonplace. Even your knowledge and your motives as a physician are questioned.

Understandably, these views have created myths. We think it's time to examine them in the light of all the facts and ramifications.



MYTH: There are no differences in quality and performance between brand-name products and their generic counterparts. The corollary is that there are no differences among products made by high-technology, quality-conscious, research-based companies and those made by commodity-type suppliers.

FACT: The Food and Drug Administration does a good job in monitoring a generally excellent drug supply. Still, it has nowhere near the resources to guarantee the quality and bioavailability of all marketed products at any given time. Just a few months ago, for example, it noted that batches of tetracycline HCl capsules which met official monograph requirements were

not bioequivalent to a reference product. As you know, there is substantial literature on this subject affecting many drugs, including such antibiotics as tetracycline and erythromycin. The record on drug recalls and court actions affirms strongly that there are differences among pharmaceutical companies and their products. Research-intensive companies have far better records than those that do no research and may practice minimum quality assurance.

MYTH: Industry favors only "expensive" brand names and denigrates all generics.

FACT: PMA companies make 90 to 95 percent of the drug supply, including, therefore, most of the generics. Drug nomenclature is not the important point; it's the competence of the manufacturer and the integrity of the product that count.

Matters.

MYTH: Generic options almost always exist.

FACT: About 55 percent of prescription drug expenditure is for single-source drugs. This means, of course, that for only 45 percent of such expenditure, is a generic prescribing option available.

MYTH: Generic prescriptions are filled with inexpensive generics, thus saving consumers large sums of money.

FACT: Market data show that you invariably prescribe—and pharmacists dispense—both brand and generically labeled products from known and trusted sources, in the best interest of patients. In most cases the patient receives a proven brand product. Savings from voluntary or mandated generic prescribing are grossly exaggerated.

MYTH: Drugs account for a major portion of the rise in health care costs.

FACT: Drugs represent a very small part of such costs. The amount of the health care dollar spent for prescription drugs was about 12 cents in 1967; today it is about 8 cents. And you as a physician are most conscious of how drug therapy can cut hospitalization, avert surgery, reduce office visits and keep patients on the job.

MYTH: Government intrusions into the marketplace will save tax money.

FACT: Government schemes always cost the taxpayer something, and the costs often exceed the benefits. Certainly, any federal “help,” such as lists of wholesale drug prices sent to all physicians and pharmacists, will be no exception. Just think of the expense of keeping them current! Moreover, wholesale prices are poor guides to actual transaction prices and even worse guides to retail prices.

The PMA Position

We believe your freedom to prescribe, either by generic or brand name, should be totally unabridged. Otherwise, your prescribing prerogatives and your relationships with patients will be seriously impaired.

The maker does matter

After the myths about price and equivalency have been shattered, one fact stands out more clearly than ever: *The maker does matter.* As always, your best guide to drug therapy for your patients is to select products—both brands and generics—from manufacturers with credentials and performance records you have come to respect.



Pharmaceutical Manufacturers Association
1155 Fifteenth Street, N.W.
Washington, D.C. 20005

REPORT OF THE SECRETARY-TREASURER

The Secretary-Treasurer attended the quarterly meetings of the Council, meetings of the Executive Commission, except on February 16, 1979.

At intervals throughout the year, the Secretary-Treasurer conferred with the Executive Secretary in matters relating to the financial status and the fiscal planning of the Association.

From time to time, relevant material was presented to the Council for consideration.

Working with the Executive Secretary, the annual budget was prepared and submitted to the Executive Commission for subsequent review by the Budget Committee in the House of Delegates.

As a representative to the Advisory Committee to the Health Professions Loan Fund of the Board of Regents, the Secretary-Treasurer participated in telephone conferences and a meeting of that group during the past year.

Respectfully submitted,
Joseph N. Hamm, M.D.
Secretary-Treasurer

The Reference Committee reviewed the report of the Secretary-Treasurer and recommended its approval as submitted.

REPORT OF THE CHAIRMAN OF THE COUNCIL

The Council of the South Dakota State Medical Association has met quarterly during the past year, having had meetings in June following the annual meeting, then in October, January and April.

The minutes of these meetings are recorded in the South Dakota Journal of Medicine.

The Council meetings have been well attended by the respective Councilors of each medical district, and I appreciate the hard work that each Councilor has put into the responsibilities of Council membership.

Responsibilities increase each year with decisions made by the Council concerning Medical Association concerns, and I feel that the Council has carried out these responsibilities very well.

Respectfully submitted,
Bruce C. Lushbough, M.D.
Chairman of the Council

The Reference Committee reviewed the report of the Chairman of the Council and recommended its approval as submitted.

REPORT OF THE AMA DELEGATE

The South Dakota State Medical Association was represented at the AMA Annual Meeting in St. Louis in June 1978 and the Interim Meeting in Chicago in December 1978 by AMA Delegate W. R. Taylor, M.D., Alternate Delegate Gerald Tracy, M.D., President of the State Medical Association Russell Harris, M.D., and Robert D. Johnson, Executive Secretary.

Comprehensive reports on each meeting have been sent to each member of the State Medical Association and will not be repeated in this report.

Your delegation is anticipating representing you at the Annual Meeting in Chicago in July 1979 and the Interim Meeting in Honolulu in December 1979. Reports to each member will follow these meetings.

It has been a pleasure serving as your Delegate.

Respectfully submitted,
W. R. Taylor, M.D.
AMA Delegate

The Reference Committee reviewed the report of the AMA Delegate and recommended its approval as submitted.

REPORT OF THE ALTERNATE AMA DELEGATE

The Alternate Delegate attended all but one Council meeting during the year and attended both of the annual AMA meetings. The continued enlightenment of the functioning of the American Medical Association and our value as individual representatives to that body becomes more abundantly apparent with each meeting.

It has been a pleasure to work with the AMA Delegate and the officers of the Medical Association and the Council during the past year.

Respectfully submitted,
Gerald E. Tracy, M.D.
AMA Alternate Delegate

The Reference Committee reviewed the report of the AMA Alternate Delegate and recommended its approval as submitted.

REPORT OF THE SPEAKER OF THE HOUSE

During the past year I have attended all the meetings of the Executive Committee including several conference telephone calls.

I have also attended the meetings of the Council and participated in the deliberations. I have also attended district meetings of committees appropriate to the position.

Respectfully submitted,
Durward M. Lang, M.D.
Speaker of the House

The Reference Committee reviewed the report of the Speaker of the House and recommended its approval as submitted.

REPORT OF THE COUNCILOR AT LARGE

As Councilor at Large, I attended most of the council meetings, the Executive Committee meetings, and some of the conference calls. I am very happy to report to the members of the South Dakota State Medical Association that I feel the problems before the Medical Associations are being very appropriately handled. I compliment the attentiveness of the members of the council and the chairmen of the commissions and their commission members for their work of this past year.

As Councilor at Large, I was appointed to represent the South Dakota State Medical Association on a search committee for the Vice President of Health Care and the Dean of the University of South Dakota Medical School. The President of the Board of Regents was at our initial meeting, and she stressed that it is in the statute and the intent of the legislature and the Board of Regents to continue the support of our four year medical school, and especially make it strong in the primary care areas with special emphasis on family practice. The President of the University of South Dakota, President Lien, I think is very aware of the influence of the South Dakota State Medical Association on the continuing success of the University Medical School. I felt that this was reflected by other members of the board of the search committee. The search committee has a fifteen man board representing the various departments of the Medical School plus other outside interested parties. Robert Bartron, M.D. is also on this board representing the Board of Regents, as is Joe Hamm, M.D. and Bruce Lushbough, M.D.

I would also like to make the members of the South Dakota State Medical Association aware of the Medical School's need for further financial aid, and all of the doctors in the state should get involved in learning about the needs and problems of the Medical School so that we can more appropriately explain these needs to our South Dakota House of Representative members and the members of the Senate.

I also feel that the South Dakota State Medical Association, along with the Medical School, should explore the possibility to see if we would be eligible for any federal funds or grants for support of our Medical School and our residency training programs.

Respectfully submitted,
James E. Ryan, M.D.
Councilor at Large

The Reference Committee reviewed the report of the Councilor at Large and recommended its approval as submitted.

REPORT OF THE EXECUTIVE COMMISSION

The Executive Commission met several times this past year to discuss business of the Association between Council meetings.

In June, the Executive Commission met under the guidance of President Ryan to consider an application received from a community for a National Health Service Corps physician. The District Medical Society of this area had approved the request and because time was short, it was handled through the Executive Commission and the application approved.

A discussion was then carried on reviewing several suggestions for projects which might provide additional income to the Association without increasing the dues at this time. It was felt that increasing accounting services for District Medical Societies should be implemented and other programs were to be investigated for their feasibility.

The next commission meeting was in August, by a conference call, to discuss implementation of the HEW second opinion consultation program in the state of South Dakota. The report of the ad hoc committee that had reviewed this matter was received. Considerable discussion followed. It was the feeling of the Commission that second opinions have always been available to patients in South Dakota through the usual channels of referral by local physicians by request to a District or State Medical Association, and it was the opinion of the Executive Commission that a federally funded program in South Dakota would not bring about improvement in the quality of medical care for the people of our state, but would add substantially to the cost of medical care for all South Dakotans. The Executive Commission approved a motion that the South Dakota State Medical Association decline participation in this program and that the sentiments be forwarded to the secretary of HEW. It was understood that this was not to preclude any other organization from submitting a proposal to carry out this program.

The next Executive Commission meeting was in September and followed a joint meeting that was held with the South Dakota Hospital Association. At that time, Doctor Hamm indicated that the School of Medicine would be re-applying for a federal grant for an AHEC in South Dakota. This grant has subsequently been approved, and the establishment of an AHEC (Area Health Education Committee) is presently in progress in South Dakota, with Rapid City being selected as the first site.

The Commission at this time again discussed second surgical opinions and the consensus was to submit a proposal to HEW for this purpose. A subsequent proposal was ultimately submitted by the Foundation for Medical Care and was turned down by HEW because they declined to finance the establishment of such a program.

The Commission then heard a report concerning testimony by an employee of Blue Cross of Sioux City, Iowa at the LRC interim hearings on cost containment that was felt to be directly derogatory to the medical profession and, since the testimony was being given in South Dakota, derogatory to the physicians of South Dakota. A letter was to be sent to Sioux City Blue Cross expressing the Association's dis-

pleasure with what was considered inaccurate and uncalled for statements by an employee of that organization.

A communication from the Secretary of Health was discussed and an appropriate reply was to be forthcoming.

The next Executive Commission meeting was a combined meeting with the Liaison Committee of our Association meeting with representatives from the School of Medicine. The school's present financial status, status of Maternal and Child Health Program, the school's Outreach Program as it pertains to continuing medical education, Allied Health Programs, grants solicited by the medical school, status of capitation grants and other problems or topics of mutual interest were discussed. It was planned that following this meeting, in the near future, the Liaison Committee and the committee from the medical school would meet with the State Health Department to discuss these and other topics of mutual concern. This meeting was held and the report of the Liaison Committee was given to the Council at its April meeting.

The last Executive Commission meeting to be reported here was held in April and the following things were discussed:

First, the 1979-80 budget was reviewed, modified and approved for presentation at the annual meeting. The chairman of the Internal Affairs Commission, Doctor Finney, was in attendance for this meeting. A discussion was held concerning a cost containment conference, and it was felt that this should be delayed for the time being because of the continuing dialogue that has gone on concerning this issue and the joint activity with the voluntary effort that has been ongoing as well.

A discussion was then had concerning further meeting with the Hospital Association, and this was deferred for specific action at a later date.

Dr. Paul Hohm was nominated for another term on the Board of Directors of the South Dakota Health Systems Agency.

A request from Milbank for a National Health Service Corps physician was reviewed and approved.

An informational discussion was carried on concerning the status of Blue Shield as it relates to the makeup of its Board of Directors and the national push from government to remove Medicare contracts from those Blue Shield organizations that have a majority of physicians on their Board of Directors. No action was taken. Further discussion of this issue will occur at the annual meeting.

A brief discussion was then carried out concerning the new Laetrile law and its relationship to the ethical practice of medicine. It was the consensus that the specific issue would be addressed at such time as any problem arose.

Respectfully submitted,
Russell H. Harris, m.d.
Chairman
Executive Commission

The Reference Committee reviewed the report of the Executive Commission and recommended its acceptance as submitted.

REPORT OF THE EXECUTIVE SECRETARY

Throughout this past year Dr. Harris and I have both sincerely enjoyed and appreciated the opportunity to meet with many of the members of the South Dakota State Medical Association at their local District Medical Societies. I cannot emphasize strongly enough the importance of the involvement of each and every physician in his District Medical Society activities and State Medical Association activities; for it is the grassroots physicians in our state whom the Medical Association attempts to represent. The Association will be able to respond to the needs and the desires of the physicians when through active participation they make known the areas of greatest interest and concern

to them. It is also through active participation in local medical society affairs that organized medicine gains its greatest strength.

The many committees and commissions of the South Dakota Medical Association found 1978-1979 to be a most active year. The chairmen and the members of these committees have spent numerous hours on behalf of the entire profession and the public of South Dakota, and for their efforts they certainly deserve our heartfelt thanks. These committees and commissions have directed a great deal of their time during 1978-1979 evaluating future problems facing organized medicine and seeking out positive solutions to these problems. It is hoped that this approach will give medicine greater lead time to react to the changing times and will also give the profession an opportunity to positively address issues rather than reacting in a crisis atmosphere in a negative fashion. Throughout the year there were several examples of how this posture was implemented. Negotiations with the Department of Social Services in obtaining a new and acceptable claim form and eliminating an offensive provider agreement is certainly one sterling example. Another example is the efforts made on behalf of the Association prior to the legislative session in working with the Board of Nursing and the Nurses Association to obtain legislation defining the scope of practice of nurse practitioners and nurse midwives. Even though it appeared early on that the Medical Association and nursing might very well have strong differing opinions on the legislation, after several hours of negotiation, an acceptable bill was drawn and passed by our legislature. The Association also established, in conjunction with the South Dakota Hospital Association, a Voluntary Effort Committee to review and make recommendations on methods to contain hospital costs on a voluntary basis rather than the mandatory approach presently being considered by the administration in Washington. These are only a few of the examples of this strategy in action, and many other times it was utilized with equal effectiveness by other commissions and committees.

I would like to take this opportunity to thank the many physicians who serve on committees of other organizations who have given very freely of their time on behalf of organized medicine. Our physician members who serve on the Joint Practice Commission, the South Dakota Foundation for Medical Care Board, the Board of Medical and Osteopathic Examiners, the SoDaPAC Board of Directors, the A.H.E.C. committees, the HSA and its sub-area councils and its many planning committees, and the Liaison Committee have continued to make certain that medicine's views are articulately expressed and considered in the deliberations of allied organizations. I would also be most certainly remiss if I did not express our gratitude to our AMA delegation who has very effectively presented the views of South Dakota physicians to our national organization.

The 1979 legislative session again found many bills with direct and measurable effects on the medical profession in South Dakota. Because our Association had requested and received a great deal of legislative time in some of the immediately preceding sessions, the Legislative Commission and the Council made a concerted effort to limit the number of areas in which our Association would become involved. However, try as we might, there were still sixteen individual bills which we followed. It would not be appropriate to reiterate each of these bills individually since they have been previously discussed in the "Grab Bag", and Dr. Haas has done an outstanding job of outlining the activities of the Commission on Legislation and Governmental Relations in a separate report. It should be noted, however, that during this legislative session we found it necessary once again to introduce legislation to help further alleviate problems with professional liability insurance. The Legislative Commission and the Council did draft and caused to have introduced a bill which provides for a two year statute of limitations on

physicians' professional corporations, a loophole which had previously not been discovered. This bill was successful during the session and will hopefully have continuing positive effects on the professional liability climate in our state. It is also worthy to note that during this session a measure was introduced which would have repealed the statute of limitations for lawyers, and although never formally introduced, an amendment was discussed which would have included physicians and other professionals in this repeal. Opposition to this move was successful. Hopefully, your Association will not have to continually defend the very positive steps taken by prior legislatures in addressing the professional liability problem.

It is my firm belief that the medical school will need renewed support of the profession in order to see the fulfillment of its goals and objectives. It became increasingly clear during this legislative session, with money being very tight and the needs of higher education being very great, that without our continued support the medical school may be unable to obtain the necessary funding to maintain the high quality program which has been established.

To your officers and councilors who have so freely given of their time and talent, I extend my grateful appreciation for it is their advice and counsel which continues to establish the positive direction for medicine in South Dakota. To Dr. Harris, your president, a very special commendation for a job well done. Dr. Harris not only gave freely of his time and talents but represented medicine in a truly exemplary fashion before lay groups, professional groups and the legislature.

Respectfully submitted,
Robert D. Johnson
Executive Secretary

The Reference Committee reviewed the report of the Executive Secretary and recommended its approval as submitted. The Committee would like to recognize the outstanding work and ability of our Executive Secretary, Robert Johnson and his staff in their continued exemplary performance during the last year in conducting the South Dakota State Medical Association business.

REPORT OF THE FIRST DISTRICT COUNCILOR

The Aberdeen District Medical Society held its regular monthly meetings from September 1978 through May 1979. Spouses were generally in attendance for a combined social hour and dinner; then held their own auxiliary meeting while the district business meeting was conducted.

First meeting of the business year was held on September 6, 1978. The scientific program was presented by Robert Raszkowski, M.D. of the University of South Dakota who discussed "Antibiotic Induced Colitis." Discussion was held regarding provider agreements of the South Dakota Department of Social Services.

The October meeting was a brief social event due to inability of the scheduled speaker to meet with us.

The State Medical Association President, Russell Harris, M.D., and Executive Secretary, Robert Johnson, met with the district on November 11, 1978 and discussed current problems concerning the Medical Association in South Dakota. Doctors Welge, Bachmayer and Mazurek were accepted into membership in the Association.

The annual meeting was held on December 6, 1978. The following officers were elected: President, Dr. A. J. Janusz; Vice President, Dr. John Christopher; Secretary/Treasurer, Dr. Barry Welge. Delegates elected were Dr. P. A. Leon, Dr. A. J. Janusz, Dr. J. C. Rodine. Alternate delegates elected were Dr. C. L. Voge, Dr. G. H. Steele, and Dr. D. Seaman.

The February meeting consisted of a tour of the 3M Plant in Aberdeen together with a dinner and brief business meeting. Doctors K. A. Bartholomew, J. R. O'Connor and

R. J. Lynn were accepted to membership.

A March meeting featured a presentation by Dr. Ismael Unite of Aberdeen concerning radionuclide scanning.

Dr. Robert Talley of the University of South Dakota School of Medicine made a presentation on pulmonary embolism for the April 4 meeting.

Dr. M. Thomas Stillman of Hennepin County Medical Center will present a discussion of arthritis therapy at the meeting of May 2.

Respectfully submitted,
B. C. Gerber, M.D.
Councilor, First District

The Reference Committee reviewed the report of the First District Councilor and recommended its acceptance as submitted.

REPORT OF THE SECOND DISTRICT COUNCILOR

The following are the activities of the District Medical Society:

April 1978—Regular meeting plus presentation by Dr. Blanton Bessinger, Pediatric Cardiologist, from the University of Minnesota in reference to the "Innocent" Murmur.

May 1978—Instructions to the delegates were given for the State Medical Association meeting as well as a scientific program by Dr. John D. Barker, Jr., M.D., Gastroenterologist, from Sioux Falls on "Acid Peptic Disease, Its Diagnosis and Treatment".

September 1978—Regular district business plus a recommendation from our district to request a National Health Service Corps psychiatrist to be affiliated with the Lake Region Mental Health Center was approved. A scientific program was presented by Dr. and Mrs. Alpers from Germany giving a brief summary and review of the practice of medicine as it exists in Germany today.

October 1978—Recommendations from the district that members of the district visit the Boys and Girls Clubs and review their facilities, particularly as it relates to physical exercise programs. A program was given by Dr. Heupel on "Rhogam".

November 1978—Communications regarding continued medical education, Central Plains Clinic, and other sources were read. There was also a discussion about the Estelline Hospital in reference to a U. S. Public Health Service, National Health Service Corps physician for their community. It was recommended by our district that this be referred to the HSA. It was at this meeting that our State President, Dr. Russell Harris, and the Executive Secretary of the State Medical Association, Mr. Robert Johnson, gave presentations and update as to the present activities and physicians of the South Dakota State Medical Association.

December 1978—Our guest was Dean Spartz, fourth year medical student in family practice rotation. At this time the new officers were elected for the District Medical Society for the coming year. They included: President Dr. Nick Guddal; Vice President Dr. B. J. Desai; Secretary Dr. Gerald Tracy; Censor for three years Dr. C. J. Clark; Delegate for two years Dr. James Rud; Alternate Delegate for Dr. Rud was Dr. Wrage for two years; Utilization and Review Committee Dr. David Piro for three years. Dr. Fuller and Dr. Arbs were to have presented a program entitled "Concepts in Pain Management"; however, this had to be cancelled at a late hour because of transportation problems.

January 1978—Regular business was conducted with guests Sister Jean Canora, Sister Del Rey, Rev. Val Sauer and Dean Spartz, medical student. A program on pastoral care for hospitals was presented by Sister Jean, Sister Del Rey, and Rev. Val Sauer.

February 1978—In addition to the regular monthly meeting, Dr. McBride gave a presentation on "Pharmacology and Therapeutics for Treatment of Hypertension". Guests were Dr. John Whitney, Medical Student IV, and Chuck

McCarthy from Smith Kline and French.

March 1978—Regular monthly district business meeting plus Dr. Fred Lovrien, Endocrinologist from Central Plains Clinic in Sioux Falls, gave a presentation on "Thyroid Function Testing".

Respectfully submitted,
G. R. Bartron, M.D.
Councilor, Second District

The Reference Committee reviewed the report of the Second District Councilor and recommended its acceptance as submitted.

REPORT OF THE THIRD DISTRICT COUNCILOR

The Third District Medical Society meetings have been held during the past year as follows:

April 20, 1978—Brookings, South Dakota—Dr. Arthur Mollen, Phoenix, Arizona spoke on the subject of "Exercise and Its Benefits".

May 25, 1978—Madison, South Dakota—The Third District recommended Bruce Lushbough, M.D. as Councilor for the next term and A. A. Lampert, Jr. M.D. as Alternate Councilor. Discussion for resolutions for the House of Delegates meeting was then carried out.

August 3, 1978—Arlington, South Dakota—Social meeting was held without a medical program.

September 27, 1978—Brookings, South Dakota—Official visitation of Russell Harris, M.D., President of the South Dakota State Medical Association, was made. Robert D. Johnson, Executive Secretary of South Dakota State Medical Association, also gave his annual presentation.

December 7, 1978—Flandreau, South Dakota—Third District meeting was held and election of officers for the year of 1979 was held.

President—Dr. Ronold Tesch
Vice President—Dr. D. L. Scheller
Secretary-Treasurer—Dr. Richard Wake
Censors—Dr. C. S. Roberts, Dr. Saul Friefeld,

Dr. Joe Muggly
Delegates—Dr. A. A. Lampert, Jr., Dr. Werner Klar
Alternate Delegates—Dr. Curtis Wait, Dr. Homer Stensrud

Respectfully submitted,
Bruce C. Lushbough, M.D.
Councilor, Third Medical District

The Reference Committee reviewed the report of the Third District Councilor and recommended its acceptance as submitted.

REPORT OF THE FOURTH DISTRICT COUNCILOR

The annual meeting of the Fourth District Medical Association was held on January 16, 1979. Officers elected were Barbara K. Spears, M.D., President; Hubert Werthmann, M.D., Vice President; M. R. Cosand, M.D., Secretary-Treasurer. Delegate to the State Association was M. R. Cosand, M.D. and Alternate Delegate was L. C. Askwig, M.D.

The program consisted of the annual visitation of Dr. Russell Harris, State Association President, and a discussion of the upcoming legislation affecting medicine by Bob Johnson, Executive Secretary.

The other district activity for the year was a cooperative effort of all the Pierre physicians in holding athletic physicals on two evenings for all of the Hughes and Stanley County athletes.

Respectfully submitted,
R. C. Jahraus, M.D.
Councilor, Fourth District

The Reference Committee reviewed the report of the Fourth District Councilor and recommended its acceptance as submitted.

REPORT OF THE FIFTH DISTRICT COUNCILOR

Our first meeting since my last report was held in Huron on May 24, 1978. The main program concerned the June meeting, councilor's report, and the instruction of the delegates to the meeting.

On October 4, 1978 Dr. Russell Harris of Rapid City spoke as President reporting on the recent activities of the State Association. New members recognized at that time were Dr. Myron Fahrenwald and Dr. Thomas Dean.

Dr. Fred Lovrien of Sioux Falls spoke on his topic of "Thyroid Function Tests" on November 30, 1978. Memorial money was sent to the State Medical Endowment Fund in memory of the death of Dr. Robert A. Buchanan of Huron.

At the last meeting held on March 29, 1979 three new members were announced: Dr. George Nicholas, Dr. Julie Kurch, and Dr. Robert Pelegrin. The program for this meeting consisted of a presentation by Psychologist Ron Goldsmith of the local Community Counselling Service. His topic was "Child Abuse".

Respectfully submitted,
David J. Buchanan, M.D.
Councilor, Fifth District

The Reference Committee reviewed the report of the Fifth District Councilor and recommended its acceptance as submitted.

REPORT OF THE SIXTH DISTRICT COUNCILOR

The Sixth District held only three meetings in 1978. Dr. Joy Taggart from Corsica and her husband were guests at the May 4th meeting. It was voted to raise the Sixth District dues to \$50.00 per year for each member. It was decided that an attempt would be made to schedule speakers from the School of Medicine or elsewhere for continued medical education. At the election of officers, it was unanimously approved that the previous year's officers be reappointed. Dr. Charles Monson and Dr. W. P. Baas were elected delegates to the state meeting in June. Alternates were Dr. W. W. Weatherill and Dr. J. O. Mabee.

Respectfully submitted,
R. G. Gere, M.D.
Councilor, Sixth District

The Reference Committee reviewed the report of the Sixth District Councilor and recommended its acceptance as submitted.

REPORT OF THE SEVENTH DISTRICT COUNCILOR

The Medical Society met monthly except during the summer and discussed topics pertinent to the membership. These include such subjects as the emergency room coverage and surgical and urgent care centers.

Respectfully submitted,
Respectfully submitted,
John F. Barlow, M.D.
Councilor, Seventh District

The Reference Committee reviewed the report of the Seventh District Councilor and recommended its acceptance as submitted.

REPORT OF THE EIGHTH DISTRICT COUNCILOR

In the past year, four meetings of the Eighth District Medical Society were held.

On March 2, 1978, significant business included resolutions to the State Medical Society supporting adequate funding for the State Human Services Center and also a resolution for the State Medical Association to support a study group to look into the possibility of vocational rehabilitation at the Human Services Center. New members accepted into the Eighth District Medical Society included Michael Mc-

Vay, M.D., Vernon Hermesen, M.D., David Bean, M.D., Paul Finninger, M.D., Fe' Cabuso, M.D., Gabriel Martino, M.D., C.C. Pascale, D.O., and L. P. Mills, D.O.

On May 17, 1978, the state president, James Ryan, M.D. visited the Medical Society. He was accompanied by the executive secretary, Bob Johnson. There was active discussion of topics of interest to physicians in South Dakota including government regulations and malpractice. New officers were elected and included president, John Willcockson, M.D.; vice-president, Phil Blum, M.D.; secretary, Hal Fletcher, M.D.; and treasurer, Jay Hubner, M.D. New members were approved and included Tom Olson, M.D., Ken Hunt, M.D., Bill Dendinger, M.D. and Phil Blum, M.D.

On September 22, 1978, a scientific program was presented. John Edmonson, M.D. of Mayo Clinic gave a program on the "Oncological Treatment of Breast Carcinoma." At the business meeting, the Area Education Center concept was discussed and the Eighth District Medical Society passed a resolution to participate and support this concept. Dr. Tom Johnson was nominated and accepted to be our representative with the development of AHEC.

On December 6, 1978, the Eighth District Medical Society had a scientific program presented by Dr. Alan Kind from the St. Louis Park Medical Clinic, who discussed the management of Cram negative infections. The business meeting was short and no significant business was concluded on this date.

Respectfully submitted,
Frank Messner, M.D.
Councilor, Eighth District

The Reference Committee reviewed the report of the Eighth District Councilor and recommended its acceptance as submitted.

GREETINGS

FROM



JERRY MAGINN

Medical Service Representative

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B. F. Ascher & Co., Inc.

Kansas City, Missouri

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President's Page

The spectre of picking up your daily newspaper in the near future and reading a headline "South Dakota Medical School Closed", is closer to reality than we wish to admit. So once again we are faced with a battle to preserve that which we fought so hard to create only four years ago. Unless adequate funding is forthcoming, the Medical School could lose its accreditation.

It is interesting to pause and consider the effect on South Dakota should the state lose the Medical School. The immediate concern of most would be the loss of potential young physicians who would provide medical care across the state. Sixty-five native young people are chosen for each incoming class. Since most medical schools across the country are tending to educate only people from their own state, it is conservatively estimated that approximately 60 of our young people would never be accepted to any medical school, thus disenfranchising them from becoming a physician, not because they were not qualified to pursue a degree in medicine, but only because they, unfortunately, were citizens of South Dakota! What a waste of talent this would be.

Should we allow the Medical School to close, we, as practitioners in this state, would lose a myriad of services that benefit us and our patients. Among these are included Basic Science Outreach programs such as visiting faculty presentations, coronary care training unit, basic coronary care courses, CPR courses and continuing education materials. The Medical School also conducted and planned over two hundred CME programs in the last year alone! Add to this the Medical Audio-Visual Services, the Lommen Health Sciences Library, and all of the consultative and referral services provided by the faculty, and one readily begins to see the impact the medical school has as a spin-off from educating our native sons and daughters.

We must remind the Veterans of our state what the degree granting school has done for them. Prior to the grant, there was no medical education program at the Sioux Falls VAMC; no housestaff, and few continuing education opportunities for profes-

sional staff existed. While good patient care was available, diverse specialized care could not be offered readily. Since the affiliation, the Sioux Falls VAMC has established itself as a clinical training site for upper division medical students. **The graduate residency program, nonexistent in FY 1975, now has 15 housestaff physicians in University based residency programs.** Specialized program expansion has included the establishment of an intensive care/coronary care unit capability, as well as the development of a nuclear medicine service and a respiratory care center. The core staff of highly trained physicians representing many subspecialties has been recruited as a result of the affiliation with the Medical School. How many of these would remain at the Sioux Falls VAMC should the Medical School close is purely a guess, but the space of specialties would, undoubtedly, erode over a period of time.

The Indian population of our state is also one that has benefited greatly by the creation of a four-year medical school. The improved prenatal and perinatal care of the Indian population in the Rosebud and Pine Ridge areas would not be possible had not the four-year medical school been created.

Too often we concern ourselves with the amount of money it costs to run the Medical School not realizing that there are many financial benefits to our state. Many thousands of dollars comes into our state as a result of grants written and supervised by our faculty. Our faculty has a payroll of over \$2,000,000 and our career service people, of which there are 91, have a salary of nearly \$1.8 million. It has been estimated by economists that a payroll dollar in our state is spent some seven different times. We could then state that the payroll alone has somewhere in the neighborhood of a \$25,000,000 impact on the state economy.

There are many other reasons for retaining the Medical School in its present form. I urge all of the Association membership to continue their support of the Medical School and continue to remind your patients and your legislators of the importance of adequate funding of this most important part of our higher education institution.

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Net wt 1/32 oz (approx)



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Each gram contains: Aerosporin[®] (Polymyxin B Sulfate) 5,000 units, bacitracin zinc 400 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base), special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

INDICATIONS: *Therapeutically*, (as an adjunct to systemic therapy when indicated), for topical infections, primary or secondary, due to susceptible organisms, as in: infected burns, skin grafts, surgical incisions, otitis externa; primary pyodermas (impetigo, ecthyma, sycosis vulgaris, paronychia); secondarily infected dermatoses (eczema, herpes, and seborrheic dermatitis); traumatic lesions, inflamed or suppurating as a result of bacterial infection. *Prophylactically*, the

ointment may be used to prevent bacterial contamination in burns, skin grafts, incisions, and other clean lesions. For abrasions, minor cuts and wounds accidentally incurred, its use may prevent the development of infection and permit wound healing.

CONTRAINDICATIONS: This product is contraindicated in those individuals who have shown hypersensitivity to any of its components. Do not use in the eyes or in the external ear canal if the eardrum is perforated.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control

secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.



SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS
3001 South Holly Avenue
Sioux Falls, SD 57105



What Should You Expect From Staff & Staff From You*

The most successful associations are those that maintain a careful balance between board and staff lines of authority and responsibilities. Yet this is an area that can create some of the most frequent misunderstandings.

As an association leader, have you ever asked yourself, "What do I expect from the association staff," and on the other hand, "What should the staff expect from me?"

The board-staff relationship should never disintegrate into an "us against them" battle. Yet, how do you maintain the delicate balance?

Here are some pointers suggested by Barnes & Roche, Inc., of Rosemont, Pennsylvania, a firm that provides consulting services in the areas of development and fund raising. These pointers apply equally well to many types of voluntary organizations.

What Should Volunteers Expect of Staff?

1. A volunteer should expect to have the time he is freely giving used to its best advantage, not wasted on things others can do or on relatively unimportant tasks.
2. A volunteer should expect to receive from a staff member options and recommendations for activity, not "what should we do about this?" or "how should we handle this?"
3. A volunteer should expect the staff members to adhere to points of view the staff believes are professionally correct, notwithstanding the attitude of volunteers. At the same time, a volunteer should expect a staff member to be flexible and to incorporate the views of volunteers into the program as appropriate.
4. A volunteer should view the staff member as a valued professional colleague who is neither dictated to nor put on a pedestal.
5. A volunteer should expect the staff to identify his role and activities and to provide all necessary background information to carry out his responsibilities.
6. A volunteer should expect to be told bad news or problems (as well as good news) and should be involved, when appropriate, in developing solutions.
7. A volunteer should expect from a staff member polite but firm pressure to achieve objectives and to move forward according to an agreed-upon plan.

What Should Staff Expect of Volunteers?

1. A staff member should expect from volunteers creativity in addressing the program and issues and not a "rubber stamp" performance. At the same time, the staff member should expect the volunteers to respect his judgment and—by and large—to accept his recommendations.
2. A staff member should expect that volunteers (no matter how involved and committed) need continuing education. Every staff contact with a volunteer should tell him more about the organization and its programs and objectives.
3. A staff member should neither fear nor denigrate his volunteers, but rather view them as valued colleagues.

4. A staff member should expect volunteers to ask hard questions.

5. A staff member should expect volunteers to have individual strengths and characteristics which must be identified and put to special use.

6. A staff member should be sensitive to the personal and business priorities of volunteers which may impinge on their ability to serve. The staff member must keep asking for help and work—with sensitivity—while being certain that the things being asked for are substantive and important.

7. A staff member should remember that more good volunteers are lost because too little, rather than too much, is asked of them.

Society of Teachers of Family Medicine Fact Sheet

The Society of Teachers of Family Medicine is an organization of teachers of the family practice discipline at both the graduate and undergraduate levels of medical education.

The Society provides an organizational medium to: (1) monitor, improve and set new standards of excellence in family practice education; (2) conduct research in teaching methods for the delivery of patient care; (3) integrate and enhance harmony among the various disciplines which are engaged in family practice; (4) promote the development of more teachers of Family Medicine; and (5) develop a good inter-relationship with other organizations in family practice.

STFM was founded in 1967. There are more than 1,400 members from the education side of family practice. Family physicians who teach at medical schools and in residency training programs, both at university medical centers and teaching community hospitals, are a part of the society. Its membership is also composed of behavioral scientists engaged in teaching non-medical aspects of family practice and community medicine; social workers who teach and function at the residency level; and other related health care professionals, such as nurse practitioners and nutritionists. The STFM membership is international in scope, as it includes members from Canada, England, and Australia.

National headquarters for the Society are at 1740 West 92nd Street in Kansas City, Missouri. STFM sponsors two academic conferences each year—a freestanding one in the spring, and the other integrated with the annual conference of the Association of American Medical Colleges in the fall. The annual membership meeting is held each year in conjunction with the free-standing spring conference of the Society.

STFM functions by means of a Board of Directors, six standing committees (Communications, Constitution & Bylaws, Education, Membership, Program and Research), and a headquarters staff. It is represented in the AAMC through membership in the Council of Academic Societies.

Annual dues for STFM are \$75 for physicians, \$50 for non-physicians, and \$10 for residents who may become associate members.

REPORT OF THE NINTH DISTRICT COUNCILOR

The Black Hills District Medical Society held six meetings during the past year. The meetings were held on May 11, 1978, September 26, 1978, November 9, 1978, December 12, 1978, January 27, 1979 and February 8, 1979. All meetings were held in Rapid City, South Dakota.

Officers for the current year are as follows:

President—Donald Kelley, M.D.

Vice President—Lee Ahrlin, M.D.

Secretary/Treasurer—A. J. Barrett, M.D.

During the year, the Black Hills District named Dr. R. L. Ferrell of Rapid City to complete the unexpired Councilor term of William Jones, M.D.

At our regular business meetings, programs were presented on a variety of topics and routine business matters were handled.

Respectfully submitted,

A. J. Barrett, M.D.

Councilor, Ninth District

The Reference Committee reviewed the report of the Ninth District Councilor and recommended its acceptance as submitted.

REPORT OF THE TENTH DISTRICT COUNCILOR

The Rosebud District Medical Society held two meetings during the past year. One of these meetings was the presidential visit of Dr. Russell Harris who was accompanied by the Executive Secretary, Bob Johnson.

Three new physicians have located in the district the past year. They are: John Malm, M.D., Gregory; Louis Hogrefe, M.D., Gregory; and Sidney Wechsler, M.D., Martin.

New officers for the district are:

President—Don Bailey, M.D., O'Neill, Nebraska

Vice President—Louis Hogrefe, M.D., Gregory

Secretary-Treasurer—E. P. Sweet, M.D., Burke

Respectfully submitted,

R. L. Stiehl, M.D.

Councilor, Tenth District

The Reference Committee reviewed the report of the Tenth District Councilor and recommended its acceptance as submitted.

REPORT OF THE ELEVENTH DISTRICT COUNCILOR

During the 1978-1979 year, the Northwest Medical District revised, approved and adopted a complete set of by-laws. During the year, there were eight monthly meetings at which there were guest speakers covering a variety of topics for at least one hour of CME credits for each meeting. Officers chosen for the 1978-1979 year were as follows:

President—Dr. Ben Henderson

Secretary/Treasurer—Dr. Leonard Linde

11th District Councilor—Dr. James Wunder

The meeting on April 20, 1979, will be for election of officers for the 1979-1980 year. A topic of consideration for the coming year will be accreditation through the University of South Dakota for CME credits for the monthly district medical meetings.

Respectfully submitted,

James F. Wunder, M.D.

Councilor, Eleventh District

The Reference Committee reviewed the report of the Eleventh District Councilor and recommended its acceptance as submitted.

REPORT OF THE TWELFTH DISTRICT COUNCILOR

On July 19, 1978, the Whetstone Valley District Medical Society met at The Galley in Webster. Dr. J. A. Eckrich, Jr.,

urologist, Aberdeen, South Dakota, spoke on testicular diseases and treatment.

On October 19, 1978, the Whetstone Valley Medical Society met in Sisseton, South Dakota. The President of the South Dakota State Medical Association, Dr. Russell Harris, was unable to attend due to illness and the Executive Secretary, Robert D. Johnson, delivered an address on current Medical Association business and problems.

The Whetstone Valley District Medical Society lost one long-time member in death, Dr. Joseph Lovering, on September 7, 1978.

Another member, Dr. Glen Oey moved to Watertown to practice there.

The next meeting of the Whetstone Valley District Medical Society will be held in Milbank on April 13, 1979.

Respectfully submitted,

Eldon E. Bell, M.D.

Councilor, Twelfth District

The Reference Committee reviewed the report of the Twelfth District Councilor and recommended its acceptance as submitted.

REPORT OF THE COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS

Two meetings of the commission were held during the course of the year. The first of these was on Saturday, September 9, 1978.

The commission reviewed and reaffirmed opposition to the Hospital Rate Review Law if it was to be introduced in the 1979 session.

Consideration was also given to problems with optometry legislation. Action regarding this was deferred at that time pending comments and response from the Ophthalmology Society.

Another major point of discussion during the course of the year was the Nurse Practice Act and Physician Assistant Law. It was the feeling of the commission that if medicine were to be practiced that it should fall under the jurisdiction of the Board of Medical and Osteopathic Examiners as opposed to Nursing Practice, which fell under the Nursing Board. At the initial meeting, a commission was appointed to further study this and to attempt to determine what the nursing profession intended or wishes with regard to this.

The Statute of Limitations Bill for medical corporations was reviewed and arrangements made to have this introduced into the 1979 legislature.

Letters from Blue Shield regarding reimbursement and from Dr. E. H. Heinrichs regarding legislation relating to child care were reviewed and accepted for information only. Blue Shield had indicated opposition to reimbursement for independent physician extender services not under a physician's supervision.

The proposed increase in licensure fees was also supported by the commission.

Additionally, a meeting was held with the members of the Department of Social Services from Pierre. Review of the Title XIX Program and contemplated changes were carried out. The discussion with these people from the Department of Social Services proved quite enlightening, particularly with reference to their support for many physicians' problems. There are apparently considerable difficulties for the department relating firstly to funding and secondly to attempting to satisfy the federal bureaucracy. It was noted that current Title XIX funding, as of September 1, 1978, was just raised to the 1975 level based on 1975 fees. Additionally, review of the new computer service contemplated to be installed for the department was also discussed. It was suggested that this would save the Department of Social Services approximately \$20,000 annually. The savings are somewhat artificial, however, since the majority of the savings comes from an increase in the

percentage federal matching grants. The total cost of running the computer program will be considerably higher than with previous methods. It does make retrievable data, however, much easier.

The Medicaid Provider Agreements out last year were reviewed. Real concerns of the physicians as to reference to the Rehabilitation Act and the uncontrolled audit of the provider by Medicaid agents were raised. It was indicated that many physicians did not feel that they could in good conscience sign this. Apparently, a number of these had not been signed, and the Department of Social Services had been continuing to pay on the basis of the old agreements previously signed. Subsequently, the approach used was to discontinue all provider agreements on the basis of yearly contracts and have the agreement based on the individual voucher signature.

Questions regarding Medical School funding and residency funding were deferred because of lack of information regarding needs at that point.

The commission also discussed a letter from the Georgia Medical Association requesting support for a constitutional amendment limiting federal deficit spending. The commission did recommend the State Medical Association endorse the concept.

The second meeting of the Commission on Legislation and Governmental Relations was held on Wednesday, December 13, at the Howard Johnson Motor Lodge in Sioux Falls.

The Commission on Nurse Practitioners and Physicians Assistants and Mr. Johnson had worked with the Nursing Board. The Nursing Board had agreed that nurse practitioners should be licensed and be under the jurisdiction of the Board of Medical and Osteopathic Examiners with reference to the medical care provided as nurse practitioners. It was also agreed supervision for nurse practitioners should be essentially the same as physicians assistants. They also concurred with a decision that supervision should be limited to two nurse practitioners. This essentially represented the agreement on every point we had raised earlier.

Proposed bills of concern for the coming legislative session were reviewed at this meeting. The commission endorsed the bill to rescind the requirement for premarital syphilis serologic testing. The proposed restraint law for children under age five was accepted for information only. The pending optometry bills were again reviewed. We had still not received any information from the Ophthalmology Society regarding their position on these bills, and a motion was made to take no position pending their recommendations. The Residency Funding Bill was endorsed. No position was taken at that time regarding the sunset for the Basic Science Board.

The proposal to change driver's license regulations for persons with seizures, requested by a Yankton area attorney, was tabled at the meeting.

The program for catastrophic illness was referred to the commission for study and recommendation.

As a result of the meeting, recommendations were to sponsor bills regarding the statute of limitations for corporations and a statute allowing necessary medical treatment for minors without parental consent.

Endorsed bills included changes in the Medical Practice Act, increased fees for the Board of Medical Examiners, and eliminate separate osteopathic examination aside from the regular medical examination for the osteopaths.

The budget for South Dakota Human Services Center and the South Dakota School of Medicine budget requests were endorsed.

Opposed bills included Hospital Rate Review Law, expansion of Physician Assistant Law to allow four physicians assistants under one employer, legalization of laetrile, statute requiring physicians to furnish medical records to chiropractors, and statute allowing chiropractors to perform high school physical examinations.

Additionally, review of the Medicaid arrangement with regard to claim forms was updated at this meeting. Physicians who had signed the new Provider Agreement were allowed to cancel that agreement with the Department of Social Services and to be reimbursed on the basis of the old Provider Agreement. As mentioned in the previous program, they expressed an intent to cancel both agreements with agreement on an individual basis based on the claim forms.

Respectfully submitted,
Stephen N. Haas, M.D.
Chairman
Commission on Legislation and
Governmental Relations

The Reference Committee carefully reviewed the report of the Commission on Legislation and Governmental Relations. The Reference Committee recommends the acceptance of the report of the Commission on Legislation and Governmental Relations.

REPORT OF THE COMMISSION ON MEDICAL SERVICE

The commission's first meeting was held September 9, 1978 at the Holiday Inn Downtown, Sioux Falls, South Dakota. A quorum was present for conduct of business and also in attendance were Dr. Russell Harris and Dr. Steven Haas. It was moved that the minutes of the previous meeting be accepted as published, and the motion was seconded and carried.

A discussion of the Emergency Medical Services Department Service Plan Draft was had and again centered around difficulties encountered in obtaining reports in return of questionnaires from hospitals in the state. It was urged that the commission members and council members bring these problems to the attention of the hospital administrators and chiefs-of-staff in their areas. Following discussion, it was moved by Dr. Warren Jones that the commission recommend to the Council that the State Medical Association accept the Emergency Medical Services draft plan. The motion was seconded and carried.

As a second order of business, there was considerable discussion concerning implementation of the Advanced Life Support Programs of the Emergency Medical Services Department. Since the grant request had been denied by the federal government, it was felt that reasons for the denial should be discussed. Members of the commission felt that there were several concerns that should be expressed to the Emergency Medical Services Advisory Committee; namely #1—the concern of hospital administrators concerning categorization of hospitals may limit or close hospitals, especially those in the small communities; #2—radio communication network was hampered by the lack of technicians to keep them in operating condition; #3—to express the concerns of the physicians that suggestions in the drafted plan may become regulations with no latitude for individual change; and #4—request an alternative if the EMS implementation plan is rejected.

The next item of business concerned the South Dakota Farm Safety Council. It was the commission's feeling that the State Medical Association should continue its membership and that two physicians, one from east river and one from west river, should be appointed to attend meetings and participate in Council activities. The appointment of Dr. Curtis Wait as East River Representative and Dr. Anthony Javurek as West River Representative were made at the time of the commission meeting.

The next order of business was the prescribing practices for nurse practitioners. Mr. Johnson reported the Attorney General had released an opinion which would not allow nurse practitioners to prescribe under the current nurse practice act. He also stated that the Health Manpower Sub-physician Sub-committee recommended that nurse practi-

tioners come under the jurisdiction of the South Dakota State Board of Medical and Osteopathic Examiners rather than the Nurses' Board. This was submitted for the commission's information only.

The next order of business reviewed the guidelines for transfer of high risk newborn as presented by the Emergency Medical Services Program. It was the commission's feeling that the guidelines as submitted were not acceptable since there was no available statistical studies indicating that these protocols were relative or warranted in the state of South Dakota, that there was no latitude for physician judgment regarding transfer of patient, and the protocols required that too many patients be transferred including both infant and mother.

In the next order of business, the Children's Comprehensive Health Care Service Program, formerly Crippled Children's Program, was discussed with Stanley Graven, M.D., and in particular, a case where a physician was notified that his services would no longer be covered by the program for a particular case inasmuch as he was not a board certified orthopedic surgeon. Dr. Graven indicated that these decisions are made by the Advisory Committee which includes four practicing physicians and that this particular case will be discussed at the next Advisory Committee meeting. Dr. Graven then outlined the South Dakota perinatal program including the statistical data utilized to apply for the federal grant and the development of the program to date. Following a rather lengthy discussion, the commission indicated to Dr. Graven that it was their hope that any new programs or projects will be brought to the attention of the commission at the time of planning rather than after the fact so that practicing physicians will have input into these programs. Dr. Graven then gave background information of the Neonatal Nurse Clinician Program which is operated through the South Dakota State University in Brookings. It was the feeling of the commission that nurses with a specialty training are needed in hospital perinatal nurseries, but recommendations from physicians concerning this program were completely ignored. The information concerning this program was submitted to the commission for information only. No action was taken.

The meeting adjourned at 1:15 p.m.

The spring meeting of the Commission on Medical Service convened on Saturday, March 31, 1979, at the Ramada Inn, Sioux Falls, South Dakota. The meeting was called to order with a quorum being present. Dr. Rud moved the minutes of the previous meeting be accepted as published. The motion was seconded and carried.

The first order of business, correspondence with the Baptist Hospital in Winner, South Dakota concerning Emergency Room Categorization, was discussed. A reply had been sent to the hospital administrator by the Chairman, Dr. Saylor, supporting the emergency room standards by the JCH and indicated that in his opinion the hospital would no way be locking itself into a level of delivery of Emergency Medical Services by use of such standards. The commission concurred with the response to the Baptist Hospital in Winner and felt that no further action was indicated.

The second order of business, The Farm Safety Council, was again discussed, and Dr. Javurek reported that he was unable to attend the last meeting of the Farm Safety Council because he received notification on the date of the meeting. The upcoming AMA Rural Health Conference, which is to be held in St. Paul April 18 to 20, was discussed. Following further discussion, Dr. Javurek moved that the commission request that the council designate the AMA Rural Health Conference as an official meeting of the State Medical Association and provide funding for a physician to attend. The motion was seconded and carried. A discussion was then held concerning the Emergency Medical Services Program of the State Health Department. Since no funding for fiscal year 1980 was applied for in view of non-

compliance with federal regulations, it appears that there will be no possibility of an advance life support system in the state at this time. The commission then reviewed the proposal from Dr. Heinrichs to require immunization records for USD students. The commission felt this would indeed be helpful to physicians to provide better medical care to students not only in Vermillion but in all state institutions and private colleges as well. It was moved by Dr. Monson that the South Dakota State Medical Association endorse this proposal to require physician certified immunization records for USD students and recommend that all other state institutions and colleges be made aware of this proposal enactment. The motion was seconded and carried.

A regional research project on Burns Injuries was next considered by the commission. This was presented by the Agricultural Experimental Station of South Dakota State University. Since the study which had previously been done in Nebraska and South Dakota is very similar, and since this was not endorsed by the Hospital Association and involved extra time and paper work for personnel of the hospital, it was moved by Dr. Hollerman, seconded and carried, that the commission recommend that the council not endorse the implementation of this project for the reasons stated above.

The recommendations from the Long Range Planning Committee regarding HMOs was next discussed. A proposal to establish a panel for the purpose of gathering statistics and information concerning HMOs was not felt necessary at this time since this would be a duplication of study from a Sioux Falls group which is now already in the process of making the study because it involved federal funding, and since the council previously had rejected recommendations to apply for grant monies for the feasibility study, it was moved that the commission receive and review information from the Sioux Health Maintenance Organization Feasibility Study along with any other information that is available, and following that review, take action on the matter. The motion was seconded and carried. The commission went on record, however, to state that any infringement in the private practice of medicine will be strongly opposed by the commission. The commission then discussed two grant applications from subcommittees on Medical School Research Projects. #1—a grant application from the Allied Health Sciences, USD on "Rural Hospital Trade Patterns and Travel Costs" was felt to be a duplication of effort and time and that a great deal of this information is already available. In view of this, the commission moved and carried the recommendation that the council not endorse this grant as presented. #2—a grant application for USD for transporting of preterm newborns was felt to be important particularly to family units in rural areas. This grant was endorsed, and it was recommended that the council accept the endorsement.

The commission next reviewed the request from the Department of Social Services that Nurse Practitioners and Physician's Assistants be allowed to sign the EPSDT screening report and claim form without a physician's co-signature. The commission concluded that the NPs and PAs need direct physician supervision, that the legal aspect had not been specifically delineated, and that this activity could lead to individual private practice of NPs and PAs. Following considerable discussion, it was moved that inasmuch as this is a medical report that could involve legal aspects—on that basis—the commission recommends to the council that the South Dakota State Medical Association strongly endorse that physicians sign these forms. The motion was seconded and carried.

The commission next discussed the resolution received from the South Dakota Pediatric Society strongly opposing chiropractors performing athletic evaluations. It was moved by Dr. Hollerman that the commission recommend that the council approve the resolution as presented along with the suggestion that this resolution be distributed to all school boards and school superintendents in South Dakota. The

motion was seconded and carried.

A review of the National Health Service and Rural Health Initiative Programs were then made with Dr. Richard Baker, representative from the National Health Service Corp, and Mr. Bernard Osberg, representative from the State Health Planning and Development Office, in attendance. Dr. Baker presented the background information on these two programs and reviewed the criteria for determining the shortage areas for the placement of physicians and allied health personnel. Dr. Baker indicated that he will communicate with the State Medical Association office when applications are made for medical personnel through the NHS and RHI programs in South Dakota so that physician comments and input can be considered. It was moved that the commission recommend to the council that the Liaison Committee of the State Medical Association meet periodically with Dr. Baker to discuss National Health Service and Rural Health Initiative Programs and what effect these programs may have on existing practices in the area. The motion was seconded and carried.

A request was submitted by the South Central Community Action Program to endorse a study on the effect of National Health Service Corp manpower on the utilization of existing medical and dental practices within the area. Following discussion, it was moved by Dr. Monson that the commission recommend to the council that the SDSMA not endorse this study proposal inasmuch as this information is available from other sources and only needs to be compiled. The motion was seconded and carried on vote.

Dr. Saylor then reviewed an article from the Emergency Services Newsletter regarding motorcycle injuries and the motorcycle helmet law. Following discussion, Dr. Rud moved that the commission recommend to the council that the SDSMA endorse reinstitution of the motorcycle helmet law in South Dakota. Motion was seconded and carried.

There being no further business, the meeting adjourned at 12:00 noon.

Respectfully submitted,
H. L. Saylor, Jr., M.D.
Chairman
Commission on Medical Service

The Reference Committee reviewed the report of the Commission on Medical Service. The Reference Committee recommends the acceptance of the report of the Commission on Medical Service.

REPORT OF THE COMMISSION ON SCIENTIFIC MEDICINE

The Commission on Scientific Medicine met twice during the past year. Meetings were held on September 9, 1978 and March 31, 1979. At the fall meeting, the commission re-

viewed the continuing medical education requirement for membership in the State Medical Association. The commission recommended at that time that no change be made in these requirements.

The commission also reviewed the 1978 annual meeting and the evaluations and comments which were provided by those in attendance. The commission planned the scientific program for the 1979 annual meeting and selected speakers to present the topics. The 1979 annual meeting's format will consist of a three day schedule rather than the four day schedule which we have utilized in recent years.

At the spring meeting, the commission reviewed a brochure submitted by the State Health Department entitled "Operation Lifestyle." The commission recommended that the State Medical Association give approval to the content of the brochure.

The commission also developed a position paper on home deliveries to be used as the official position of the State Medical Association. This position paper was developed after contacting the South Dakota Ob-Gyn Society, the South Dakota Pediatric Society, the South Dakota Chapter of the American Academy of Family Physicians, and the South Dakota Nurses Association for input.

At the spring meeting, the commission again discussed the continuing medical education requirement and recommended that the implementation of this requirement be delayed for one year. This matter will be considered by the House of Delegates at the 1979 annual meeting.

Respectfully submitted,
James Larson, M.D.
Chairman
Commission on Scientific Medicine

The Reference Committee considered the report of the Commission on Scientific Medicine. The Reference Committee recommends the acceptance of the report of the Commission on Scientific Medicine.

REPORT OF THE COMMISSION ON INTERNAL AFFAIRS, COMMUNICATIONS AND LIAISON

The commission met twice during the past year. Both meetings were held in Sioux Falls. The commission recommended to the council the endorsement of the program of I. C. Systems, Inc. by the South Dakota State Medical Association.

The commission also recommended that the composition of the Budget and Audit Committee be the Executive Committee and the chairman of the Commission on Internal Affairs, Communications and Liaison.

A film was shown at the fall meeting entitled "Health Caring—From Our End of the Speculum" by the Commission on the Status of Women along with the Health Care

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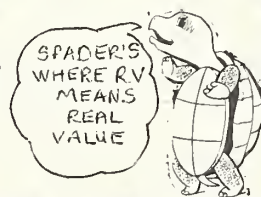


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Task Force of the Commission on the Status of Women. After review and discussion, it was the recommendation of the commission that the council give consideration to commending the Commission on the Status of Women for establishing the stated objective and trying to improve the knowledge of women on health care matters and thereby improving the health care of women in South Dakota. However, the Commission on Internal Affairs, Communications and Liaison did not feel that the stated objective would be served at all by the means selected—i.e., the showing of this film. The commission recommended that the council take appropriate action to call to the attention of the proper state bodies their concern about the showing of this film by the Commission on the Status of Women.

The commission discussed the progress on the physician recruitment program. It was the recommendation of the commission to the council that they continue the program on a modified basis and continue to make contact with physicians in residency programs.

The South Dakota State Medical Association bylaws were reviewed by the commission at its spring meeting. It was felt by the members of the commission that the present bylaws are current and needed no updating at this time.

The commission reviewed a resolution proposed by the Budget and Audit Committee to change the bylaws which would repeal the continuing medical education requirement of 150 hours every three years as required for membership in the State Medical Association. It was the recommendation of the commission to the council that this resolution be submitted to the House of Delegates.

The commission met with the president of the Women's Auxiliary and discussed fostering a closer liaison between the Women's Auxiliary and the State Medical Association with the recommendation to the council that a representative of the auxiliary be named to attend meetings of the Commission on Internal Affairs and Legislation in an effort to coordinate their efforts in the field of these two commissions.

An Avis Rent-a-Car System, Inc. car rental proposal was reviewed by the commission. It was the commission's recommendation to the council that the proposal be accepted.

Recommendations from the Long Range Planning Committee suggesting that the district society structure within the State Medical Association be reviewed were considered by the commission. It was the recommendation of the commission that in the foreseeable future, realigning of district and of state association is not practical. Most of the continuing education programs have been under hospital sponsorship. In addition, it was felt that considerable resistance in some areas would be encountered to the loss of representation to the state association.

The commission considered requests from various organizations to obtain the association's mailing list. It was the recommendation of the commission that the mailing list be made available to individual physicians, allied health organizations and commercial organizations; charges to be determined as costs are considered, a minimum of \$50 for commercial organizations.

A proposal by Northwestern Bell Telephone Company regarding directory advertising for physicians was considered, and it was the recommendation to the council that this program be referred to the council for their recommendation.

During the past year, the following South Dakota physicians have died: L. E. Savage, M.D., Yankton, died May 1978; Joseph Lovering, M.D., Webster, died September 1978; Robert A. Buchanan, M.D., Huron, died October 1978; and Paul Reagan, M.D., died April 1979.

Health Career Loan Fund reported the following activity during the past twelve months:

Balance in Savings Account	
March 9, 1979	\$19,114.27
Income	
Interest	\$ 1,497.74
Principal (Loan repayments) ..	2,527.32
Total Income	\$ 4,025.06
	4,025.06

Transferred to 6 month	
Money Market Certificates ...	\$12,000.00
Expenses	
Loans (11)	5,500.00
Total Transferred Out of Savings ..	\$17,500.00
	\$17,500.00

Balance in Savings Account	
March 10, 1979	\$ 5,639.33
Assets	
Money Market Certificate ...	\$12,000.00
Savings Account	5,639.33
	\$17,639.33
Outstanding Loans	10,230.39
	\$27,869.72

During the year, the commission has reviewed each financial report of the South Dakota State Medical Association, Journal account and the building fund. Budget and Audit Committee, consisting of the Executive Committee and the chairman of this commission, approved the budget for the fiscal year 1978-79. The budget was submitted to the council and approved for transmittal to the House of Delegates. The proposed budget is attached as part of this report.

Respectfully submitted,
Lawrence W. Finney, M.D.
Chairman
Commission on Internal Affairs,
Communications and Liaison

The Reference Committee considered the report of the Commission on Internal Affairs, Communications and Liaison. In reviewing the proposed budget for the fiscal year 1979-1980 the Commission took note of the recommendation from the Budget and Audit Committee and the Council that the in-state travel allowance for the president of the South Dakota State Medical Association be increased to \$1,000.00 per year, from \$500.00. This change would increase the item for Physician Travel to \$7,250.00 and decrease the anticipated reserve to \$3,550.00. The Reference Committee recommends that this change be approved. The Reference Committee recommends the acceptance of the report of the Commission on Internal Affairs, Communications and Liaison with this one adjustment.

PROPOSED BUDGET 1978-79 SOUTH DAKOTA STATE MEDICAL ASSOCIATION GENERAL FUND

ITEM	INCOME	
	BUDGETED 78-79	PROPOSED 79-80
State Dues	\$140,000.00	\$145,000.00
Annual Meeting	13,000.00	14,000.00
Refunds & Misc.	4,000.00	4,000.00
Car Reimbursement	250.00	250.00
Continuing Medical Education	1,000.00	1,000.00
Adminis. Reimbursement	7,200.00	7,800.00
	\$165,450.00	
Other Programs		
7th District		1,200.00
AAFP		1,200.00
Collection Service		1,000.00
Interest Income		5,000.00
WPPA		750.00
		\$181,200.00

EXPENSES		
ITEM	BUDGETED 78-79	PROPOSED 79-80
Salary, Executive Secretary	\$ 21,500.00	\$ 24,500.00
Salary, Other	57,500.00	62,500.00
Social Security	4,900.00	5,500.00
Legal & Audit	5,500.00	5,500.00
Telephone	4,000.00	3,500.00
Office Supplies	5,800.00	6,500.00
Dues & Sub.	500.00	600.00
Physicians' Travel	6,000.00	6,750.00
Annual Meeting	13,000.00	15,000.00
Public Relations	5,500.00	5,000.00
Journal Subsidy	5,000.00	5,500.00
Postage	6,500.00	4,500.00
Miscellaneous	100.00	100.00
Legislative Expense	4,200.00	3,000.00
Car Expense	1,200.00	1,200.00
Staff Travel	8,500.00	8,500.00
Insurance	1,500.00	1,500.00
Retire/Fringe Benefits	12,000.00	13,000.00
Taxes	200.00	200.00
Aux. Newsletter	800.00	800.00
Employment Tax	150.00	200.00
CME	800.00	800.00
Income Tax	—	1,000.00
Replacement of Equipment	—	2,000.00
	<u>\$165,150.00</u>	<u>\$177,150.00</u>
Reserve	300.00	4,050.00
	<u>\$165,450.00</u>	<u>\$181,200.00</u>

BUILDING FUND INCOME		
ITEM	BUDGETED 78-79	PROPOSED 79-80
Foundation Rent	\$ 10,800.00	\$ 10,800.00
Bd. of Exam. Rent	1,800.00	2,400.00
Interest Income	7,000.00	7,500.00
	<u>\$ 19,600.00</u>	<u>\$ 20,700.00</u>

EXPENSES		
ITEM	BUDGETED 78-79	PROPOSED 79-80
Salaries, Staff	\$ 8,100.00	\$ 9,500.00
Utilities	2,300.00	2,600.00
Taxes & Insurance	4,000.00	4,200.00
Maint. & Supplies	3,000.00	3,200.00
Legal & Audit	1,200.00	1,200.00
	<u>\$ 18,600.00</u>	<u>\$ 20,700.00</u>
Reserve	1,000.00	
	<u>\$ 19,600.00</u>	

JOURNAL INCOME		
ITEM	BUDGETED 78-79	PROPOSED 79-80
Advertising	\$ 16,000.00	\$ 17,000.00
Subscriptions	1,000.00	1,200.00
Refunds	720.00	720.00
Journal Subsidy	5,000.00	5,000.00
Miscellaneous	300.00	500.00
Contribution	600.00	—
	<u>\$ 23,620.00</u>	<u>\$ 24,420.00</u>

EXPENSES		
ITEM	BUDGETED 78-79	PROPOSED 79-80
Salary, Editor	\$ 720.00	\$ 720.00
Salary, Staff	1,500.00	1,500.00
Legal & Audit	100.00	100.00

Travel		
Social Security	100.00	100.00
Telephone	100.00	100.00
Postage	1,000.00	1,000.00
Office Sup. & Print.	20,000.00	20,900.00
	<u>\$ 23,520.00</u>	<u>\$ 24,420.00</u>
Reserve	100.00	
	<u>\$ 23,620.00</u>	

REPORT OF THE COMMISSION ON PROFESSIONAL LIABILITY

The last meeting of this commission was in November, 1978. Since that time, Robert Johnson and I have traveled to Omaha and had numerous discussions with Dick Bielski relative to physician owned insurance companies in the state of South Dakota.

At the November meeting, a formal presentation was made by the Nebraska Physician Owned Malpractice Corporation. Much discussion and questions followed the presentation. Bob Johnson and I were commissioned to travel to Omaha. We had lunch with the president of the corporation, one of the other officers (both physicians), and the executive secretary of the insurance corporation.

What developed is as follows:

First of all, to do nothing; to stay with St. Paul Fire and Marine as the major provider in the state of South Dakota.

Secondly, to begin the corporation proceedings and attempt to set up the South Dakota physician owned corporation.

Thirdly, to attempt to align ourselves with either the Nebraska corporation or some other existing physician owned malpractice company such as New Mexico, Arizona, or some others.

Relative to number one: At this time, the physicians in South Dakota are having neither a problem in getting malpractice insurance, nor is the price excessive to most of the insured. The possibility always exists that the St. Paul company will either increase the premiums because of the increasing malpractice problem in South Dakota, or pull out of the state altogether. A determination should be made as to the likelihood of either of those alternatives.

The second alternative is to establish a physician owned corporation in South Dakota for the purpose of providing malpractice insurance. The primary problem that I see facing us on that alternative is the requirement that we have 250 doctors as members of the corporation with a total available pool of doctors for this company of something around 500. I think a serious question must be addressed as to whether that could be accomplished. The average family practitioner's malpractice bill is less than \$2,000 a year at this time. I don't know that it would be prudent for that person to abandon St. Paul Fire and Marine, a multi-million dollar—multi-national company, to become insured by a company with no track record, i.e., the physician owned corporation. The possibility of aligning ourselves administratively with Nebraska is a plus and would certainly lower the overhead.

Lastly, the possibility of aligning ourselves with an already existing corporation. If it could be arranged, it would allow a smaller number of interested physicians the opportunity of becoming a part of a physician owned company without having to have 250 doctors signed up. (The Nebraska Physician Corporation was asked to evaluate the possibility of some doctors in South Dakota joining their corporation; they have not as yet returned to us with any information.) It was felt at the meeting in Omaha that this was not compatible with the existing Nebraska law.

In summary, my personal opinion is that with the outstanding support and commitment in South Dakota by St. Paul Fire and Marine and the present low premiums, along

with the insurmountable difficulty, in my view, of getting 250 doctors to sign up, a South Dakota physician corporation is not a viable alternative. If we are worried that St. Paul will be increasing our premiums excessively, or beginning to pull out altogether, I would suggest a serious effort be made to align ourselves with a pre-existing corporation such as Nebraska, New Mexico, etc.

Respectfully submitted,
Michael C. Rost, M.D.
Chairman
Commission on Professional Liability

The Reference Committee reviewed the report of the Commission on Professional Liability. The Reference Committee recommends adoption of this report.

REPORT OF THE COMMITTEE FOR CONTINUING MEDICAL EDUCATION

During the past year, the State Committee has inspected and approved the Continuing Medical Education Program of the Combined Medical Staff of St. Ann's Hospital—Memorial Medical Center, Watertown, South Dakota. The appropriate forms have been completed and sent to the national office for review.

A very active part in continuing medical education in South Dakota has been taken by the USD School of Medicine.

Respectfully submitted,
Gene Koob, M.D.
Chairman
Committee for Continuing Medical Education

The Reference Committee reviewed the report of the Committee for Continuing Medical Education. The Reference Committee recommends adoption of this report.

REPORT OF THE GRIEVANCE COMMISSION

Members of the committee are R. E. Van Demark, M.D., Sioux Falls; G. E. Tracy, M.D., Watertown; Fred Leigh, M.D., Huron; James E. Ryan, M.D., Mobridge; and T. H. Sattler, M.D., Yankton. The committee responded successfully to all of the various requests that required our attention.

The committee also accepted the council's charge to develop an Impaired Physician's Program for South Dakota. A formal proposal has been submitted to the council of the State Medical Association. The goal of the Impaired Physicians Program, administered by the Grievance Commission, is to assist the impaired physician in achieving maximum effectiveness, both personally and professionally.

Respectfully submitted,
T. H. Sattler, M.D.
Chairman

The Reference Committee reviewed the report of the Grievance Committee. The Reference Committee recommends the adoption of this report and recommends that when the mechanism for the Impaired Physician Program has been established, information on the program be disseminated to the members of the State Medical Association.

REPORT OF THE LONG RANGE PLANNING COMMITTEE

Members of the committee are E. H. Heinrichs, M.D., Karl Wegner, M.D., Dennis Johnson, M.D., Michael Pekas, M.D., C. E. Tesar, M.D., Homer Stensrud, M.D., W. N. Guddal, M.D., T. H. Sattler, M.D., and Russell Harris, M.D., Ex-Officio.

The committee met on July 11, 1978, November 7, 1978 and March 6, 1979. Following the guidelines established last year, the committee continued to monitor the previously-

identified primary areas of study and updated these evaluations.

All findings and recommendations were forwarded to the respective commissions and to the council of the State Medical Association.

The constant monitoring of the HSA remains a primary responsibility and concern for this committee. The committee commends the physicians of the South Dakota State Medical Association for their commitment to the work on the HSA Committees and Task Forces and their essential contribution to help develop issue papers and final health plan development for South Dakota. It remains very essential that members of the Medical Association continue to responsibly participate in developing the best possible state health plan.

One additional area for identified study was addressed. The importance and expected major impact of the Area Health Education Centers (AHEC) on the health care system needs to be fully appreciated by all members of the Medical Association. Therefore, it was urged that the AHEC project be presented in detail to all of the Medical Association members at the earliest opportunity.

Complete reports are on file with the council and the State Medical Association office.

Respectfully submitted,
T. H. Sattler, M.D.
Chairman

The Reference Committee reviewed the report of the Committee on Long Range Planning. The Committee noted for clarification that the AHEC will be supervised by the School of Medicine and encourages physicians in South Dakota to be involved in the implementation of this program. The Committee also recommended the Commission on Scientific Medicine consider including a session on AHEC for the 1980 annual meeting and that the Councilors be encouraged to bring information on this project to the attention of physicians in their districts. The Reference Committee recommended adoption of this report.

REPORT OF THE SOUTH DAKOTA POLITICAL ACTION COMMITTEE

1978 was a great year for SoDaPAC. Although we fell a shade short of our highest membership achieved in 1977, we did raise more money than ever before. As a result, we were able to have a much greater impact on legislative races than ever before.

SoDaPAC ranked first in the nation in dollars raised per member and second in ratio of sustaining members to total membership.

A great deal of the credit for such a successful year should go to the Auxiliary and its representation on our Board of Directors. I dare say we had greater Auxiliary activity and support than any other state PAC.

As you know, we are a bipartisan organization, reflected in the party affiliation of those supported in state legislative races. We feel strongly that this posture strengthens our effectiveness. Candidate support is granted only after thorough discussion and debate and evaluation of specific candidate backgrounds.

We hope we have convinced more people of the effectiveness and importance of the PAC movement in medicine so that we can have even greater support of SDSMA and Auxiliary members in 1979 and future years.

Respectfully submitted,
T. J. Wrage, Jr., M.D.
Chairman

The Reference Committee reviewed the report of the South Dakota Political Action Committee. The Reference Committee recommends adoption of this report.

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S D Laboratory Aids

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Serum Ferritin

Ferritin is the major iron storage protein in humans. It is a high molecular weight compound with a hollow shell structure which binds ferrous ions as ferric hydroxide. It is manufactured by the liver and is measured in the serum by radioimmunoassay. The levels vary with sex and age but reflect the amount of body iron stores comparing results with sensitive but complex gastrointestinal radionuclide iron absorption techniques and more common assays of serum iron, transferrin and percent saturation of transferrin. Serum ferritin levels also correlate well with estimations of bone marrow iron stores as indicated by iron stain of bone marrow aspirates. It is certainly hoped that serum ferritin determination could replace bone marrow aspiration and staining as a method of evaluating iron stores. It should also be pointed out that bone marrow staining also does not always reflect iron stores in certain patients such as young children. Serum ferritin may be helpful in this regard.

Decline in serum ferritin is an earlier indicator of iron deficiency than the serum iron or iron binding capacity (transferrin level) and occurs before alteration of the red cell indices or anemia. Measurement of ferritin may also be of help to differentiate iron deficiency from other causes of anemia in which the serum iron is decreased or the percent saturation of transferrin gives a borderline value.

Serum ferritin is often markedly elevated in iron overload states but does not totally reflect iron stores at very high levels of iron storage. However, the serum ferritin will decrease as the iron stores are decreased therapeutically.

Since normal levels vary with age and sex, pertinent information must be indicated by the physician referring the test so that appropriate normals can be ascertained.

Until measurement of serum ferritin there has not been a practical noninvasive measurement to monitor body iron stores in guiding iron therapy. We hope that excessive inadequate or inappropriate iron therapy is avoided with the measurement of serum ferritin.

John F. Barlow, M.D.
Pathologist

QUALITY HEALTH CARE

AT THE LOWEST POSSIBLE COST

The Blue Shield Association with the participation and advice of medical specialty groups such as: the American College of Physicians, the American College of Radiology, the American College of Surgeons, and the American Academy of Family Practice, has developed a program to reduce the incidence of medical procedures which contribute to cost without a parallel contribution to the quality of care.

While the Medical Necessity Program is the responsibility of the Blue Shield Association, these specialty groups working in their respective areas of practice have voluntarily assisted in the programs, by helping identify procedures which are, in most circumstances, of dubious current usefulness.

The following is a list of those procedures already identified:

Bronchoscopy—with injection of contrast medium for bronchography

Bronchoscopy—with injection of radioactive substance

Ligation of internal mammary arteries, unilateral

Ligation of internal mammary arteries, bilateral

Radical hemorrhoidectomy, Whitehead type, including removal of entire pile bearing area

Omentopexy for establishing collateral circulation in portal obstruction

Kidney decapsulation, unilateral

Kidney decapsulation, bilateral

Perirenal insufflation

Nephropexy: fixation or suspension of kidney (independent procedure), unilateral

Circumcision, female

Hysterotomy, non-obstetrical, vaginal

Supracervical hysterectomy: subtotal hysterectomy, with or without tubes and/or ovaries, one or both

Uterine suspension

Uterine suspension, with presacral sympathectomy

Ligation of thyroid arteries (independent procedure)

Hypogastric or presacral neurectomy (independent procedure)

Angiocardiography, single plane, supervision and interpretation in conjunction with cineradiography

Angiocardiography, multi-plane, supervision and interpretation in conjunction with cineradiography

Angiocardiography, utilizing CO₂ method, supervision and interpretation only

Angiography—coronary, unilateral selective injection supervision and interpretation only, single view unless emergency

Angiography—extremity, unilateral, supervision and interpretation only, single view unless emergency

Protein bound iodine (PBI)

Icterus index

Basal metabolic rate (BMR)

Phonocardiogram with interpretation and report, and with indirect carotid artery tracing or similar study

Ballistocardiogram

Fabric wrapping of abdominal aneurysm

Extra-intra cranial arterial bypass for stroke

Fascia lata by stripper for lower back pain

Fascia lata by incision and area exposure, with removal of sheet for lower back pain

Ligation of femoral vein, unilateral or bilateral for post-phlebitic syndrome

Excision of carotid body tumor, with or without excision of carotid artery for asthma

Sympathectomy, thoracolumbar, unilateral or bilateral for hypertension

Sympathectomy, lumbar, unilateral or bilateral for hypertension

Splanchnicectomy, unilateral or bilateral for hypertension

We do not recommend that physicians categorically discontinue these procedures. Almost every procedure can be medically justified in a specific instance. We do recommend, however, that each physician determine whether the results of any procedure justify the cost.

HEALTH CARE COST CONTAINMENT IS EVERYONE'S RESPONSIBILITY



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1601 WEST MADISON
SIOUX FALLS, SOUTH DAKOTA 57104
605-336-1976**

REPORT OF THE ENDOWMENT ASSOCIATION

The Endowment Association met at the annual meeting, has conducted its business throughout the year, and our financial report will not be available until the time of the annual meeting. The past year has been reasonably successful as physicians continue to contribute to the Endowment Association. The Endowment Association has also embarked on a new program as a joint venture with the University of South Dakota in hiring a full time secretary for the Alumni Association and in participating in the development and formation of an Alumni Association with the hope of building a greater support for the Medical School through the development of a solid alumni foundation. It is hoped that this will not deter from the Endowment Association and its overall attempts to help the Medical School in any possible way, but that it might also provide the tremendous support which is needed for the four year Medical School. Further reports will be forthcoming from both the Endowment Association and the Alumni Association.

The staff of the Medical Association which has helped with the Endowment Association this year, as always in the past, is to be congratulated on the fine work as well as the secretary of the Endowment Association.

Respectfully submitted,
Gerald E. Tracy, M.D.
Chairman
Endowment Association

The Reference Committee reviewed the report of the Endowment Association. The Reference Committee recommends the adoption of this report.

ANNUAL MEETING MINUTES SOUTH DAKOTA MEDICAL SERVICE, INC. CORPORATE BODY MEETING

June 8, 1979, 9:40 a.m.

Howard Johnson Motor Lodge, Rapid City, SD

Chairman Aspaas called the meeting of the Corporate Body of South Dakota Medical Service, Inc., to order at 9:40 o'clock a.m. on June 8, 1979, in the Jefferson Room of the Howard Johnson Motor Lodge in Rapid City, South Dakota.

Upon roll call, the following members of the Corporate Body of the South Dakota Medical Service, Inc., were present:

Doctors Russell Harris, Duane Reaney, Winston Odland, Joseph Hamm, Durward Lang, Wm. Taylor, Gerald Tracy, James Ryan, G. Robert Barrton, Bruce Lushbough, R.C. Jahraus, David Buchanan, R.G. Gere, Paul Aspaas, D.G. Ortmeier, Wm. Rossing, Frank Messner, Gordon Held, A.J. Barrett, N.R. Whitney, James Wunder, Eldon Bell, Paul Leon, A.J. Janusz, James Rud, James Larson, Joseph Muggly, Werner Klar, Charles Monson, Walter Baas, Guy Tam, L.J. Hyland, D.L. Johnson, R.O. Wyatt, L.W. Finney, Wm. Dendinger, Robert Neumayr, Lee Ahrlin, C.E. Tesar, Thomas Mead, Wm. Jones, M. George Thompson, David Yecha, E.A. Johnson, L.C. Askwig, E.A. Hofer, Gail Benson, Thomas Henry, Tim Zoellner.

Also present were Ex-officio non-voting Medical School Delegates:

Stewart Abbot, Student Councilor
T. Holm, Student Delegate
Randy Hart, Student Delegate

A quorum being present, the Chairman declared the annual meeting of the Membership of the Corporate Body of South Dakota Medical Service, Inc., to be duly in session for the transaction of business.

Dr. Gerald Tracy moved that reading of the minutes of the last meeting of the Corporate Body, being the 1978

Annual Meeting, be waived, the same having been published in the Corporate Handbook previously mailed to each member. Such motion was seconded by Dr. James Larson. Upon voice vote, the same was approved unanimously.

Dr. Aspaas, Chairman of the Board, presented the Chairman's message to the Corporate Body. He referred to new developments during the past year such as proposed Federal Trade Rules on Board composition of Blue Shield Plans throughout the country; Second Consultation Programs initiated by HEW; and regionalization efforts being promoted throughout the country and on which the South Dakota Blue Shield Board went on record as opposing. Dr. Aspaas further referred the delegates to his written report to the membership as contained in the Delegate's Handbook.

Dr. Aspaas referred the delegates to the financial report included in the Delegate's Handbook.

Dr. Hamm asked how many subscribers in South Dakota had medical coverage through South Dakota Blue Shield. Mr. Erickson reported that 21% of South Dakotans were covered privately by Blue Shield contracts. Another 12% of the population are covered by Blue Shield by its operation of Medicare.

Mr. Erickson noted that South Dakota Blue Shield in 1978 paid out 85.4% of each premium dollar on claims. About 12.3% of premium dollars went for administration.

Dr. Harris asked what percentage of commercial insurers' premium dollars went for expenses. Mr. Erickson stated he thought that about 28%, and that generally speaking commercial insurance companies paid out about 65% of premium dollars on medical claims.

Chairman Aspaas called for approval of the financial report of South Dakota Medical Services, Inc., for the year of 1978. Dr. Bruce Lushbough moved that the financial report be approved as presented. Dr. Tracy seconded such motion. Upon voice vote, the same was approved unanimously.

The Chairman called for the report of the Nominating Committee. The Chairman of the Nominating Committee submitted the following persons' names for nominations for re-election to the Board:

Donald Howe of Spearfish, South Dakota,
Judson O. Mabee, M.D. of Mitchell, South Dakota;
Robert Van Demark, M.D. of Sioux Falls, South Dakota;
Dennis G. Ortmeier, M.D. of Sioux Falls, South Dakota.
He reported that all four of the persons nominated are eligible for a three-year term.

The Chairman called for nomination by members of the Corporate Body from the floor for offices of Blue Shield directors. No nominations were made from the Body.

Dr. Mead moved that the nominations cease and that the Secretary be instructed to cast a unanimous ballot for the nominees, namely Donald Howe, Judson O. Mabee, M.D., Robert VanDemark, M.D., and Dennis G. Ortmeier, M.D. Such motion was seconded by Dr. Tracy. Upon voice vote, such motion was approved unanimously.

The Chairman called for consideration of Old Business. There being none, he called for consideration of any New Business.

Dr. Lushbough pointed out that in certain group plans doctors that deliver babies are not paid for newborn care in the hospital. Mr. Erickson reported that Blue Shield does not allow a fee for both delivery and newborn care of a well baby. Mr. Erickson reported that well baby care, whether by the same physician or a second specialist, is not covered. Sick baby care is paid whether to the delivering obstetrician or to an additional specialist.

Mr. Erickson referred them to a recent article printed in the Wall Street Journal relative to Blue Shield Plans and the Federal Trade Commission relative to doctors on Blue Shield Boards.

The Chairman requested other New Business. Dr. Hamm stated that he believes the financial report of South Dakota

Blue Shield shows a substantial improvement during the past year and it shows the Board and its management have done an excellent job of keeping administration expenses down and that the Corporate Body should congratulate the Board and the administrators of Blue Shield. He moved that the House of Delegates recognize the contribution of the lay members on the Board, the work of the doctors on the board and the administrative staff for what they have done in operating Blue Shield. The motion was seconded by Dr. David Buchanan. Upon voice vote, this was approved unanimously.

Dr. Aspaas thanked the Corporate Body for their cooperation on Blue Shield Programs throughout the past year and the assistance that they have always given South Dakota Blue Shield.

There being no further business, Dr. G.E. Tracy moved that the meeting be adjourned. Such motion was seconded by Dr. Bruce Lushbough. Upon voice vote, the same was approved unanimously.

The meeting was duly adjourned at 10:15 o'clock a.m.
John H. Zimmer
Secretary

PRESIDENTIAL OATH OF OFFICE

I SOLEMNLY SWEAR THAT I shall carry out the duties of the President of the South Dakota State Medical Association to the best of my ability. I shall strive constantly to maintain the ethics of the medical profession and to promote the public health and welfare. I shall dedicate myself and my office to improving health standards and to the task of bringing increasingly improved medical care to the people of South Dakota. I shall uphold the Constitution and Bylaws of the AMA and the South Dakota State Medical Association. I shall champion the cause of freedom in medical practice and freedom for all my fellow Americans.

I do solemnly swear that I will discharge the duties of this office to the best of my ability, so help me God.

DISTINGUISHED SERVICE AWARD

Started in 1951—T. F. Riggs, M.D., Pierre (deceased)
1952—H. Russell Brown, M.D., Watertown (deceased)
1953—Guy Van Demark, M.D., Sioux Falls (deceased)
1954—J. C. Ohlmacher, M.D., Vermillion (deceased)
1955—R. G. Mayer, M.D., Aberdeen (deceased)
1956—J. C. Ohlmacher, M.D., Vermillion (deceased)
1957—W. E. Donahoe, M.D., Sioux Falls (deceased)
1958—Drs. J. C. Hagin (deceased), M. W. Pangburn (deceased), and James DeGeest, Miller
1958—J. F. Brenckle, M.D., Superior, Wisc. (deceased)
1958—Mrs. Agnes Holdridge, Madison
1959—Walter L. Hard, Ph.D., Vermillion
1959—Rev. and Mrs. Robert O. Bates, Sturgis
1959—R. M. Kilgard, M.D., Watertown (deceased)
1960—L. J. Pankow, M.D., Sioux Falls (deceased)

1961—Gregg M. Evans, Ph.D., Custer
1962—Edward Shaw, Ph.D., Vermillion (deceased)
1963—Arthur A. Lampert, M.D., Rapid City
1964—John C. Foster, Phoenix, Arizona
1965—A. P. Reding, M.D., Marion
1966—Mrs. C. Rodney Stoltz, Watertown
1967—Mrs. William Fish, Watertown
1968—G. J. Bloemendaal, M.D., Ipswich
1969—F. W. Haas, M.D., Yankton (deceased)
1970—Paul Bunker, M.D., Aberdeen (deceased)
1971—E. T. Lietzke, M.D., Beresford (deceased)
1972—C. B. McVay, M.D., Yankton
1973—G. E. Tracy, M.D., Watertown
1974—J. A. Muggly, M.D., Madison
1975—Harvey Wollman, Hitchcock
1976—R. H. Quinn, M.D., Sioux Falls
1977—E. H. Heinrichs, M.D., Vermillion
1978—John Olson, Rapid City, and Evans Nord, Sioux Falls
1979—Helen Jane Hare, M.D., Rapid City

COMMUNITY SERVICE AWARD

1961—R. A. Buchanan, M.D., Huron (deceased)
1962—Roland F. Hubner, M.D., Yankton
1963—George W. Mills, M.D., Wall (deceased)
1964—John C. Hagin, M.D., Miller (deceased)
1965—Alonzo P. Peeke, M.D., Volga
1966—Hugo C. Andre, M.D., Vermillion (deceased)
1967—G. Robert Bartron, M.D., Watertown
1968—M. M. Morrissey, M.D., Pierre (deceased)
1969—N. J. Sundet, M.D., Kadoka (deceased)
1970—W. H. Saxton, M.D., Huron (deceased)
1971—R. E. Van Demark, M.D., Sioux Falls
1972—R. H. Hayes, M.D., Wall
1973—B. F. King, M.D., Aberdeen (deceased)
1974—M. C. Tank, M.D., Brookings
1975—Karl Wegner, M.D., Sioux Falls
1976—John T. Elston, M.D., Rapid City
1977—W. F. Stanage, M.D., Yankton
1978—C. S. Roberts, Jr., M.D., Brookings
1979—C. J. McDonald, M.D., Sioux Falls

AESCULAPIUS AWARD

1966—Paul R. Leon, M.D.
Walter Miller, M.D., Aberdeen
1968—H. Phil Gross, M.D., Sioux Falls

FIFTY YEAR CLUB MEMBERS

C. V. Auld, Plankinton (deceased)
R. A. Buchanan, M.D., Huron (deceased)
John L. Calene, M.D., California (deceased)
Myrtle Carney, M.D., Ft. Worth, Texas
J. C. Clark, M.D., Sioux Falls (deceased)
F. L. Class, M.D., Huron (deceased)
M. E. Cogswell, M.D., Wolsey (deceased)

J. Cook, M.D., Bonesteel (deceased)
 Harold L. Crane, M.D., Avon, Conn. (deceased)
 S. A. Donahoe, M.D., Sioux Falls (deceased)
 W. E. Donahoe, M.D., Sioux Falls (deceased)
 V. W. Embree, M.D., Pierre (deceased)
 W. D. Farrell, M.D., Aberdeen (deceased)
 R. B. Fleeger, M.D., Lead (deceased)
 R. R. Fisk, M.D., Flandreau (deceased)
 F. W. Freyberg, M.D., Mitchell
 E. E. Gage, M.D., Sioux Falls (deceased)
 D. A. Gregory, M.D., Milbank
 E. H. Grove, M.D., Arlington (deceased)
 J. C. Hagin, M.D., Miller (deceased)
 Lyle Hare, M.D., Spearfish (deceased)
 J. A. Hohf, M.D., Yankton (deceased)
 F. S. Howe, M.D., Deadwood (deceased)
 A. H. Hovne, M.D., Salem (deceased)
 A. S. Jackson, M.D., Rapid City (deceased)
 R. J. Jackson, M.D., Hot Springs (deceased)
 J. A. Jacotel, M.D., Milbank (deceased)
 G. T. Jordan, M.D., Vermillion (deceased)
 F. F. Keene, M.D., Wessington Springs (deceased)
 J. H. Lloyd, M.D., Mitchell
 O. J. Mabee, M.D., Mitchell
 P. V. McCarthy, M.D., Aberdeen (deceased)
 G. W. Mills, M.D., Wall (deceased)

B. C. Murdy, M.D., Aberdeen (deceased)
 T. F. O'Toole, M.D., Rapid City (deceased)
 N. T. Owen, M.D., Rapid City (deceased)
 L. L. Parke, M.D., Canton (deceased)
 M. O. Pemberton, M.D., Deadwood (deceased)
 R. J. Quinn, M.D., Sioux Falls (deceased)
 F. J. Radusch, M.D., California
 T. B. Ranney, M.D., Aberdeen (deceased)
 T. F. Riggs, M.D., Pierre (deceased)
 I. R. Salladay, M.D., Ft. Meade (deceased)
 W. H. Saxton, M.D., Huron (deceased)
 H. L. Saylor, M.D., Huron (deceased)
 C. E. Sherwood, M.D., Brookings (deceased)
 F. J. Tobin, M.D., Mitchell (deceased)
 Leonard W. Tobin, M.D., Mitchell
 J. S. Tschetter, M.D., Huron (deceased)
 F. W. Valkenaar, M.D., Chancellor (deceased)
 G. E. Van Demark, M.D., Sioux Falls (deceased)
 H. P. Volin, M.D., Lennox (deceased)
 C. H. Weishaar, M.D., Aberdeen (deceased)
 J. R. Westaby, M.D., Madison (deceased)
 G. E. Zimmerman, M.D., Missoula, Montana
 (deceased)
 G. J. Bloemendaal, M.D., Ipswich
 A. P. Peeke, M.D., Volga

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SD

This Is Your Medical Association

Alfred Hartmann, M.D., Sioux Falls, conducted a workshop on clinical chemistry during the joint spring meeting of the American Society of Clinical Pathologists and the College of American Pathologists in New Orleans.

* * * *

The Sioux Falls Art Auction Committee and the Civic Fine Arts Association presented the Don Douthit Award to **H. W. Farrell, M.D.**, Sioux Falls. Dr. Farrell has served on the board and as president of the Civic Fine Arts Association and the Sioux Empire Arts Council.

* * * *

Arthur Kilness, M.D., Rapid City, was inducted into the Howard Wood-Dakota Relays Hall of Fame. He was recognized for his pole vaulting abilities while attending Augustana College.

* * * *

The St. Luke's Hospital educational center in Aberdeen was designated as the **Carson B. Murdy, M.D.** Educational Center during a special meeting of the medical and dental staff. This designation was made in honor of Dr. Carson Murdy's many years of service to the hospital and the community of Aberdeen.

* * * *

Karl Wegner, M.D. was honored at the USD commencement ceremonies with a distinguished service award for contributions to the university community.

John B. Davis, M.D. has joined the Medical Associates Clinic in Pierre in the practice of general medicine. He is a graduate of the University of South Dakota School of Medicine and received his M.D. degree from the University of Alabama. He served his internship at Sacred Heart Hospital, Yankton, prior to locating in Pierre.

* * * *

R. E. Van Demark, M.D., Sioux Falls participated in the annual orthopedic crippled children's clinic in Watertown, sponsored by the Watertown Elks Lodge. The clinic has been an annual event since the 1940's.

* * * *

Col. Eldon Bell, M.D., Webster, completed flight surgeon's training at the Army Aeromedical Center at Fort Rucker, Alabama. He received training in the principles of aeromedical evacuation, immediate treatment of casualties and flying safety.

* * * *

Eighteen medical assistants were pinned at the ninth annual ceremony in the Lake Area Vocational Technical Institute's courtyard, Watertown. **Lloyd C. Vogelgesang, M.D.**, Webster, was the speaker for this event.

* * * *

Mrs. P. K. (Helen) Aspaas, Dell Rapids, state president of the PEO Sisterhood, presided over the 63rd annual S. D. State PEO Convention held in Huron.

* * * *

Three Aberdeen physicians have been named to the St. Luke's Hospital long-range planning committee, following action taken by the hospital Board of Trustees. The three are **Paul Leon, M.D.**, **C. L. Vocele, M.D.** and **Carlton Kom, M.D.**

* * * *

Mrs. Robert Van Demark of Sioux Falls, past president of the South Dakota Medical Auxiliary, was in Aberdeen to induct the new district officers for the auxiliary. The Aberdeen District auxiliary officers are: **Mrs. Juan Chavier**, president; **Mrs. Tom Bunker**, vice president; **Mrs. Joe Chang**, secretary and **Mrs. A. C. Vocele**, treasurer, all from Aberdeen.

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KENNEDY'S NEW HEALTH PROPOSAL: MORE SLOGAN THAN REALITY

As their National Health Insurance plan of the year, Senator Ted Kennedy and his helpmates have built a bright-eyed new statue with the same old clay.

The new "Health Care for All Americans" bill differs from his old "Health Security" bill in various particulars but not in basic materials.

Again the public would save on health bills by paying substantially more in tax bills. By the proponents' own estimates, the new federal costs would be \$30 billion in 1983, the year targeted for implementation. And as we know, the costs of federal programs—especially social ones—have a facility for growing.

Again there would be the heavy hand of government intervention and regulation.

But instead of being squeezed under, as in the Health Security bill, it appears private health insurers would be allowed a role of subordination. Government would regulate them and preside over negotiations on their premiums and on physician and hospital payments, and no company would be "at risk" in the true insurance sense.

In the spirit of the old bill, there would be a built-in stress and strain between health care promises and economic prohibitions.

On the one hand, the measure would guarantee

comprehensive health insurance coverage to every American at all times, amplify Medicare coverage (with no deductibles or co-insurance), reform Medicaid (eliminating the means test), and so forth.

On the other hand, the program would be subject to a national budget, whereby health expenditures would be strictly limited by state and regional allocations. Providers would have to furnish all services within the limit.

If funds run out before the end of a budget period, would providers be expected to subsidize health services? Would budget restraints and negotiation of insurance premiums and provider payments invite government rationing of care?

Those are among the grim but realistic questions to be raised as Mr. Kennedy and his supporters try to supply all the answers.

Certainly we all want answers—sound answers—to present gaps and shortcomings in health protection. In the words of AMA Executive Vice President James H. Sammons, M.D., "The AMA continues to believe that consumer choice, private insurance, and limited government regulation should be at the heart of our health care system."

"Medicine again faces a renewed responsibility to speak out on the major legislative proposals dealing with health care and to share our concerns with the Congress and our fellow citizens."

South Dakota State Medical Association Roster — 1979

Membership by Districts

ABERDEEN DISTRICT No. 1

Pres., A. J. Janusz, M.D. Sec., B. G. Welge, M.D.

Albano, P.	Aberdeen	Harlow, M. C.	Aberdeen	Rodine, J. C.	Aberdeen
Altman, S.	Aberdeen	Hovland, James I.	Aberdeen	*Rudolph, E. A.	California
*Avotins, R.	Faulkton	Janusz, A. J.	Aberdeen	Sanders, M. E.	Aberdeen
Bachmayer, Jay	Aberdeen	Kom, C.	Aberdeen	Scheffel, A.	Redfield
Berg, S.	Redfield	Kosse, Karl	Aberdeen	Seaman, David	Aberdeen
*Bloemendaal, G. J.	Ipswich	Leon, Paul	Aberdeen	Shinghal, K.	Aberdeen
Broadhurst, K. A.	Aberdeen	Lynn, Robert	Aberdeen	Shinghal, P.	Aberdeen
Bunker, Thomas	Aberdeen	McFee, John L.	Ipswich	Shousha, Alfred	Britton
Carter, P. B.	Aberdeen	McGee, Robert C.	Aberdeen	Steele, G. H.	Aberdeen
Chang, Joe P.	Aberdeen	McIntosh, G. F.	Eureka	Sweeny, W. T.	Aberdeen
Chavier, Juan R.	Aberdeen	Murdy, C. B.	Aberdeen	Taylor, Wm. R.	Aberdeen
Christopher, John	Aberdeen	*Norgello, V.	Sioux Falls	Unite, I.	Aberdeen
Driver, I.	Aberdeen	O'Connor, Jeffrey	Faulkton	Vogele, A. C.	Aberdeen
D'Souza, E. P.	Aberdeen	Odland, W. B.	Aberdeen	Vogele, C. L.	Aberdeen
Eckrich, J. A.	Aberdeen	Ostrowski, Susan	Eureka	Welge, B. G.	Aberdeen
Eckrich, J. A., Jr.	Aberdeen	Patterson, D.	Redfield	Zvejnieks, Karlis	Aberdeen
Gerber, B. C.	Aberdeen	Pelton, Charles	Aberdeen		

WATERTOWN DISTRICT No. 2

Pres., W. N. Guddal, M.D. Sec., G. E. Tracy, M.D.

Allen, S.	Watertown	Guddal, W. N.	Watertown	Piro, David F.	Watertown
Anderson, D. R.	Watertown	Hanson, B.	Watertown	Rittmann, John	Watertown
Argabrite, J. W.	Watertown	Heupel, Alden R.	Watertown	Rousseau, M. C.	Watertown
Bartron, G. Robert	Watertown	Hughes, H. D.	Clear Lake	Rud, James	Watertown
Bartron, H. J., Jr.	Watertown	*Huppler, E. G.	Watertown	Stoltz, C. R.	Watertown
Brakss, V.	Watertown	Larson, James C.	Watertown	Stransky, J. J.	Watertown
Clark, C. J.	Watertown	Meyer, Robert	Watertown	Thompson, M. C.	Watertown
Desai, B. J.	Watertown	Michieli, Jose	Watertown	Tracy, G. E.	Watertown
Fedt, D.	Watertown	Nelson, P. S.	Watertown	Wrage, T. J., Jr.	Watertown

MADISON-BROOKINGS DISTRICT No. 3

Pres., Ronald Tesch, M.D. Sec., Richard Wake, M.D.

Anderson, J. A.	Madison	Lushbough, B. C.	Brookings	Scheller, D. L.	Arlington
Appelwick, J.	Madison	Muggly, J. A.	Madison	Shaskey, R. E.	Brookings
Friefeld, S.	Brookings	Otey, B. T.	Flandreau	Stensrud, H. J.	Madison
Henry, Robert	Brookings	Patt, W. H.	Brookings	Tesch, R.	Brookings
*Kershner, C. M.	Brookings	*Plowman, E. T.	Brookings	Wait, C.	Brookings
Klar, W.	Flandreau	Primrose, Joseph	Brookings	Wake, Richard	Brookings
Kroack, K. J.	Brookings	Reagan, J. L.	Madison	*Wold, H. R.	Madison
Lampert, A. A., Jr.	Madison	Roberts, C. S., Jr.	Brookings		

PIERRE DISTRICT No. 4

Pres., Barbara Spears, M.D. Sec., Marion Cosand, M.D.

Askwig, L. C.	Pierre	Jahraus, R. C.	Pierre	Swanson, C. L.	Pierre
Collins, E. H.	Gettysburg	Lindbloom, B. O.	Pierre	Werthman, H. E.	Pierre
Cosand, M.	Pierre	Park, Dai H.	Pierre	Zakahi, R. J.	Pierre
*Fox, S. W.	California	Spears, B.	Pierre		

HURON DISTRICT No. 5

Pres., Arlan Zastrow, M.D. Sec., Emil Hofer, M.D.

Adams, H. P.	Huron	Hofer, E. A.	Huron	Lardinois, C. C.	Huron
Bell, G. Robert	DeSmet	Hohm, P.	Huron	Leigh, F. D.	Huron
Buchanan, D.	Huron	Hohm, R.	Huron	Lenz, B. T.	Huron
Dean, Roscoe	Wess. Springs	Hohm, T.	Huron	Monfore, James	Miller
Dean, Thomas	Wess. Springs	Huet, G. M.	Huron	Nicholas, George	Huron
DeGeest, J. H.	Miller	Kapur, R.	Huron	Pelegrin, R. J.	Huron
Fahrenwald, Myron	Conde	Karlen, L. W.	DeSmet	Saylor, H. L., Jr.	Huron
Gryte, C. F.	Huron	Kim, Thomas	Huron	Zastrow, A.	Oregon
Hanson, Wm. O.	Huron				

MITCHELL **DISTRICT No. 6**

Pres., C. D. Monson, M.D. Sec., Christopher Moller, M.D.

Aemkopung, C. Florida
Baas, Walter Mitchell
Berry, J. T. Mitchell
Binder, C. F. Chamberlain
Delaney, Robert Mitchell
Delaney, W. A., Jr. Mitchell
Gere, R. G. Mitchell
Gillis, F. D. Mitchell
Hermann, H. T. Mitchell
Hockett, Richard Mitchell

Holland, L. W. Chamberlain
Judge, J. O. Mitchell
Lewis, H. R. Mitchell
*Lloyd, J. H. Mitchell
Mabee, J. O. Mitchell
Mabee, O. J. Mitchell
McCann, J. P. Parkston
Moller, C. Mitchell
Monson, C. D. Parkston

Mueller, E. H. Tripp
Porter, M. Parkston
Schabauer, E. A. Mitchell
Skogmo, B. R. Mitchell
*Tobin, L. W. Mitchell
Vonburg, V. R. Mitchell
Vose, J. L. Mitchell
Weatherill, D. W. Mitchell
Williams, H. S. Mitchell

SIoux FALLS **DISTRICT No. 7**

Pres., Lowell Hyland, M.D. Sec., J. A. Ochsner, M.D. Treas., Ronald Wyatt, M.D.

Aceto, T. Sioux Falls
*Alcorn, F. A. Sioux Falls
Alvine, F. G. Sioux Falls
Amundson, Loren Sioux Falls
Anderson, C. M.S.
Anderson, Edward Sioux Falls
Anderson, T. R. Sioux Falls
Anderson, W. R. Sioux Falls
Angelos, T. Canton
Arneson, W. A. Sioux Falls
Aspaas, P. K. Dell Rapids
Aspaas, Paul Jr. Sioux Falls
Barker, J. D. Sioux Falls
Barlow, J. F. Sioux Falls
Barnett, G. L. Sioux Falls
Benson, G. Sioux Falls
Bhatti, T. H. Sioux Falls
Billion, J. J. Sioux Falls
Billion, T. J., Jr. Sioux Falls
Blake, J. Sioux Falls
Boade, W. A. Sioux Falls
Breit, D. H. Sioux Falls
Brzica, S. M. Sioux Falls
Bucy, C. Beresford
Burkhart, T. Sioux Falls
Burns, E. A. Sioux Falls
Burns, K. Sioux Falls
*Carney, M. Texas
Chalmers, J. H. Sioux Falls
Church, W. G. Sioux Falls
*Cottam, G. I. W. Sioux Falls
Cutshall, V. H. Sioux Falls
Cutshall, V. K. Sioux Falls
Daw, E. F. Sioux Falls
DeClark, R. P. Sioux Falls
Devick, J. S. Colton
Donahoe, J. W. Sioux Falls
*Eirinberg, I. Sioux Falls
Ensberg, D. Sioux Falls
Entwistle, F. R. Sioux Falls
Epp, D. Freeman
Farkas, E. C. Sioux Falls
Farrell, H. W. Sioux Falls
Felker, James Sioux Falls
Ferrell, M. R. Sioux Falls
Finney, L. W. Sioux Falls
*Fisk, R. G. Dell Rapids
Flora, G. C. Sioux Falls
Friess, R. W. Sioux Falls
Frost, D. M. Sioux Falls
Fuller, Wm. C. Sioux Falls
Gehring, S. Sioux Falls
Getz-Larsen, L. Sioux Falls
Giebink, R. R. Sioux Falls
Graham, Donald Sioux Falls
Graven, S. Sioux Falls
Greenfield, D. L. Sioux Falls
Greenfield, R. E. Sioux Falls
Gregg, J. B. Sioux Falls

Groote, C. Sioux Falls
Gross, H. Phil Sioux Falls
*Grove, M. S. Sioux Falls
Gunnarson, R. E. Sioux Falls
Gutch, C. F. Sioux Falls
Hartzell, A. Sioux Falls
Henrickson, L. Sioux Falls
Hermanson, J. M. ... Valley Springs
Hoody, Howard Sioux Falls
Hosen, R. S. Sioux Falls
Hoskins, John Sioux Falls
Hoxtell, Eugene Sioux Falls
Humphreys, D. Sioux Falls
Hurley, Brian Sioux Falls
Hussain, Rif'at Sioux Falls
Hyland, L. Sioux Falls
Ingvaldstad, J. Sioux Falls
Janis, J. B. Sioux Falls
Jaqua, R. A. Sioux Falls
Johnson, D. L. Sioux Falls
Johnson, R. C. Sioux Falls
Jones, W. L. Sioux Falls
Kaufman, I. I. Freeman
Kemp, E. Sioux Falls
Kennelly, Daniel Sioux Falls
Kenny, Jean Sioux Falls
*King, L. M. Sioux Falls
Kittelson, H. O. Sioux Falls
Knudson, D. Sioux Falls
Knutson, Dennis Sioux Falls
*Kohlmeier, F. C. Sioux Falls
Koob, K. Sioux Falls
Lakstigala, P. Sioux Falls
Lang, Durward Sioux Falls
Larsen, Paul Sioux Falls
Larson, Leland J. Sioux Falls
Leander, R. B. Sioux Falls
Lee, S. G. Sioux Falls
Looby, T. Sioux Falls
Loos, G. D. Sioux Falls
Lovrien, F. Sioux Falls
Madison, Dean Sioux Falls
Magnuson, G. Sioux Falls
*Maresh, E. R. Sioux Falls
Mark, C. Viborg
McDonald, C. J. Sioux Falls
*McGreevy, John Sioux Falls
McGreevy, P. S. Sioux Falls
McHardy, B. R. Sioux Falls
McMillin, J. Sioux Falls
Munro, R. S. Sioux Falls
Munson, D. Sioux Falls
Mutch, M. G. Sioux Falls
Naughton, G. Sioux Falls
Nelson, Earl Viborg
Nelson, R. E. Sioux Falls
Nelson, Ralph Sioux Falls
Nice, Richard Sioux Falls
Nordstrom, D. Sioux Falls

O'Brien, P. Sioux Falls
Ochsner, J. A. Sioux Falls
*Ogborn, R. J. Sioux Falls
Opheim, W. L. Sioux Falls
Opheim, W. O. V. Sioux Falls
Orr, R. T. Sioux Falls
Ortmeier, Denny Sioux Falls
Parry, R. Sioux Falls
Pasek, E. A. Sioux Falls
Paxson, C. Sioux Falls
Payne, H. Sioux Falls
Peik, D. J. Sioux Falls
Pekas, M. Sioux Falls
Petereit, M. F. Sioux Falls
Peters, E. H. Sioux Falls
Petres, A. Salem
Pitt-Hart, Barry T. Sioux Falls
Quale, J. Sioux Falls
Quinn, R. H. Sioux Falls
Read, R. Sioux Falls
Regier, E. Canton
Reynolds, James Sioux Falls
Richards, George Sioux Falls
Rossing, W. O. Sioux Falls
Rost, M. Sioux Falls
Rutt, Carl Sioux Falls
Salmela, S. Sioux Falls
Sanchez, G. Sioux Falls
Sanderson, E. W. Sioux Falls
Schultz, R. D. Sioux Falls
Seidenstein, L. Sioux Falls
*Sercl, W. Sioux Falls
Shreves, H. Sioux Falls
Sittner, Larry Sioux Falls
Smith, G. Sioux Falls
*Stahmann, F. Sioux Falls
*Steiner, P. K. California
Stensland, V. Sioux Falls
Stoltz, C. R. Sioux Falls
Sweeney, L. J. Arizona
Talley, Robert Sioux Falls
Tam, Guy Sioux Falls
Thatcher, L. G. Sioux Falls
Tschetter, L. K. Sioux Falls
Tschetter, R. T. Sioux Falls
Ulus, M. Sioux Falls
Van Demark, R. E. Sioux Falls
Villa, Jose Freeman
Vogt, H. B. Sioux Falls
Volin, V. V. Sioux Falls
Waltner, Lonnie Bridgewater
Walton, J. E. Sioux Falls
Wegner, K. H. Sioux Falls
White, T. C. Sioux Falls
Wierda, D. R. Sioux Falls
Williams, B. J. Sioux Falls
Willix, Robert Sioux Falls
Wyatt, George Sioux Falls
Wyatt, R. Sioux Falls

YANKTON DISTRICT No. 8

Pres., John Willcockson, M.D.

Sec., H. J. Fletcher, M.D.

Treas., Jay Hubner, M.D.

Alonzo, Leoncio Yankton
Auld, M. A. Yankton
Bean, David Yankton
Brookman, B. T. Wagner
Dendinger, Wm. Vermillion
Fletcher, H. Vermillion
Foley, R. J. Tyndall
Halverson, K. Yankton
Held, G. Yankton
Hermesen, V. H. Yankton
*Hill, J. F. Yankton
Hollerman, Chas. Yankton
Holzwarth, D. R. Yankton
Honke, R. W. Wagner
Hubner, J. Yankton
*Hubner, R. F. Yankton
Isburg, Carroll Yankton

Jameson, G. M. Yankton
Johnson, T. C. Yankton
Johnson, V. Vermillion
Kalda, E. F. Platte
Leshar, R. Yankton
Lyso, M. Yankton
McVay, C. B. Yankton
†McVay, M. R. Kentucky
Messner, F. Yankton
Mills, L. P. Platte
Neumayr, R. J. Yankton
Olson, Thomas Vermillion
Petersen, L. Yankton
Porter, Richard I. Yankton
Price, Ronald Armour
Quick, Wm. Yankton
Radack, Morris Yankton

Ramos, M. Scotland
Ranney, B. Yankton
Reaney, D. B. Yankton
Reding, A. P. Marion
Saloum, H. Tyndall
Saoi, N. B. Yankton
Sattler, T. H. Yankton
Sebring, F. U. Vermillion
Stanage, W. F. Yankton
Steele, J. P. Yankton
Thompson, R. F. Yankton
Thornton, R. R. Yankton
Tidd, J. T. Yankton
Tuan, C. Yankton
Turner, C. R. Vermillion
Willcockson, John Yankton
Willcockson, T. H. Yankton

BLACK HILLS DISTRICT No. 9

Pres., C. E. Tesar, M.D.

Sec., A. J. Barrett, M.D.

Ahrlin, H. L. Rapid City
Ahrlin, H. L., Jr. Rapid City
Allen, Bruce Rapid City
Anderson, A. B. Lead
Bailey, J. D. Rapid City
Bareis, R. J. Rapid City
Barrett, A. J. Rapid City
Behrens, C. L. Rapid City
Bergeron, Dale Rapid City
Berkebile, Dale Rapid City
Berry, D. Sturgis
Bloemendaal, R. D. Rapid City
Blunck, C. J. Rapid City
*Borgmeyer, H. J. Rapid City
Boyce, R. A. Rapid City
Boyer, D. W. Rapid City
Branch, Robert Rapid City
*Bray, R. B. Rapid City
Brown, Michael Spearfish
Burnap, D. W. Rapid City
Burnett, R. Rapid City
Butz, Gerald Rapid City
Cameron, D. E. Rapid City
Carlson, G. Rapid City
Carson, L. E. Arkansas
*Clark, B. S. Spearfish
Cline, J. A. North Carolina
Cornford, R. C. Rapid City
Dewald, A. Rapid City
Drummond, R. Rapid City
*Dulaney, C. H. Ft. Meade
Dzintars, P. F. Rapid City
Ebbert, Larry Rapid City
Elston, J. T. Rapid City
Eulberg, Marny Hot Springs
Ferrell, R. Rapid City
Finley, R. C. Rapid City
Forshner, R. Hot Springs
Freimark, L. G. Rapid City

Fromm, H. E. Rapid City
Frost, H. L. Rapid City
Gilbert, F. J. Ft. Meade
Gollither, W. N. Spearfish
Gwinn, C. B. Rapid City
Haas, S. Rapid City
Hafner, Daniel Rapid City
Hamm, Joseph Rapid City
Hare, H. J. Rapid City
Harris, R. H. Rapid City
Haugan, H. O. Rapid City
Hayes, Robert Wall
Heidepreim, G. Rapid City
Henry, Thomas Rapid City
Hercules, C. Rapid City
Hewitt, J. M. Rapid City
Howard, Wm. J. Rapid City
Jacobson, T. R. Hot Springs
James, E. Rapid City
Janss, Gerti Rapid City
Javurek, A. J. Deadwood
Jenter, G. W. Sturgis
Johnson, Robert K. Rapid City
Jones, W. E. Sturgis
*Kegaries, D. L. Rapid City
Kelley, D. H. Rapid City
Kovarik, J. A. Rapid City
Kovarik, R. A. Rapid City
Kovarik, W. J. Rapid City
Krafka, T. Rapid City
Kunz, J. A. Rapid City
Kwan, F. P. Rapid City
Lampert, A. A. Rapid City
Loos, C. Rapid City
Lopez, A. Hot Springs
Massa, L. L. Sturgis
Mattox, J. E. Deadwood
Mattson, W. Rapid City

McGuigan, P. M. Rapid City
Mead, T. Spearfish
Merryman, M. P. Rapid City
Millea, R. P. Rapid City
Miner, J. Rapid City
Munson, H. B. Rapid City
Neu, Norman Rapid City
O'Sullivan, J. Belle Fourche
Owen, G. S. Rapid City
Palmerton, E. S. Rapid City
Perry, Wm. J. Ft. Meade
Pieczka, Jan Hot Springs
*Radusch, F. J. California
Reinoehl, W. Custer
Renka, R. Rapid City
Ruud, E. T. Hot Springs
Sabow, J. D. Rapid City
*Saxton, A. J. Arizona
Sejvar, J. P. Rapid City
Slingsby, J. B. Rapid City
Smart, E. Belle Fourche
Smith, David Deadwood
Strand, R. D. Rapid City
Swisher, L. P. Kadoka
Tesar, C. E. Rapid City
Theissen, H. H. Rapid City
Trinidad, R. Deadwood
Tschetter, W. R. Rapid City
Vogele, Kenneth Rapid City
Welsh, Gary Rapid City
Westaby, R. S. Hot Springs
Whitney, N. R. Rapid City
Wicks, Dennis Custer
*Williams, F. R. Rapid City
Wood, G. F. Rapid City
Wright, Paul Rapid City
Yackley, J. V. Rapid City
Zanka, J. A. Rapid City

ROSEBUD DISTRICT No. 10

Pres., Don Bailey, M.D.

Sec., Ed Sweet, M.D.

Bailey, Donald O'Neill, Neb.
Nemer, R. G. Gregory

Stiehl, R. Winner
Sweet, E. P. Burke

Thompson, M. George Gregory

NORTHWEST DISTRICT No. 11

Pres., R. R. Lawrence, M.D.

Sec., L. M. Linde, M.D.

Collins, J. D. Hoven
Dietrick, L. A. Bison
Henderson, B. J. Mobridge
Johnson, C. A. Lemmon
Knowles-Smith, P. McLaughlin

Lawrence, R. R. Mobridge
Linde, Leonard Mobridge
*Nolan, B. P. Mobridge
Peterson, Jeffrey Mobridge

Ryan, J. E. Mobridge
*Spiry, A. W. Mobridge
Wunder, J. Mobridge
Yecha, David Gettysburg

WHETSTONE VALLEY DISTRICT No. 12

Pres., L. F. Nelson, M.D. Sec., Eldon Bell, M.D.

Bell, Eldon Webster
*Brinkman, W. C. Sisseton
Buentipo, B. Milbank
*Gregory, D. A. Milbank

Janavs, V. Milbank
Johnson, E. A. Milbank
Kass, Joseph Rosholt
Mendoza, V. Sisseton

Nelson, L. F. Webster
Oey, D. Sisseton
Staub, D. W. Sisseton
Vogelgesang, L. C. Webster

M.S.—Indicates Military Service
*—Indicates Honorary Membership

South Dakota State Medical Association Roster — 1979 Membership — Alphabetical Listing

Aceto, T. Sioux Falls
Adams, H. P. Huron
Aemkopung, C. Florida
Ahrlin, H. L. Rapid City
Ahrlin, H. L., Jr. Rapid City
Albano, P. Aberdeen
*Alcorn, F. A. Sioux Falls
Allen, Bruce Rapid City
Allen, S. W. Watertown
Altman, S. Aberdeen
Alonzo, L. Yankton
Alvine, F. G. Sioux Falls
Amundson, Loren Sioux Falls
Anderson, A. B. Lead
Anderson, C. M.S.
Anderson, D. R. Watertown
Anderson, Edward Sioux Falls
Anderson, J. A. Madison
Anderson, T. R. Sioux Falls
Anderson, W. R. Sioux Falls
Angelos, T. Canton
Appelwick, J. Madison
Argabrite, J. W. Watertown
Arneson, W. A. Sioux Falls
Askwig, L. C. Pierre
Aspaas, P. K. Dell Rapids
Aspaas, Paul Jr. Sioux Falls
Auld, M. A. Yankton
*Avotins, R. Faulkton
Baas, W. Mitchell
Bachmayer, Jay Aberdeen
Bailey, Don O'Neill, Neb.
Bailey, J. D. Rapid City
Bareis, R. J. Rapid City
Barker, J. D. Sioux Falls
Barlow, J. F. Sioux Falls
Barnett, G. L. Sioux Falls
Barrett, A. J. Rapid City
Bartron, G. R. Watertown
Bartron, H. J., Jr. Watertown
Bean, David Yankton
Behrens, C. L. Rapid City
Bell, Eldon Webster
Bell, G. Robert DeSmet
Benson, G. Sioux Falls
Berg, S. Redfield
Bergeron, Dale Rapid City
Berkebile, D. Rapid City
Berry, D. Sturgis
Berry, J. T. Mitchell
Bhatti, J. H. Sioux Falls
Billion, J. J. Sioux Falls
Billion, T. J., Jr. Sioux Falls
Binder, C. F. Chamberlain
Blake, J. Sioux Falls
*Bloemendaal, G. J. Ipswich
Bloemendaal, R. D. Rapid City
Blunck, C. F. Rapid City
Boade, W. A. Sioux Falls
*Borgmeyer, H. J. Rapid City
Boyce, R. A. Rapid City
Boyer, D. Rapid City
Brakss, V. Watertown
Branch, R. Rapid City

*Bray, R. B. Rapid City
Breit, D. H. Sioux Falls
*Brinkman, W. C. Sisseton
Broadhurst, K. A. Aberdeen
Brookman, B. T. Wagner
Brown, M. Spearfish
Brzica, S. M. Sioux Falls
Buchanan, D. Huron
Bucy, Christine Beresford
Buentipo, B. Milbank
Bunker, T. Aberdeen
Burkhart, T. Sioux Falls
Burns, E. A. Sioux Falls
Burns, K. R. Sioux Falls
Burnap, D. W. Rapid City
Burnett, R. Rapid City
Butz, Gerald Rapid City
Cameron, D. E. Rapid City
Carlson, G. Rapid City
*Carney, M. Ft. Worth, Texas
Carson, L. E. Arkansas
Carter, P. B. Aberdeen
Chalmers, J. H. Sioux Falls
Chang, J. P. Aberdeen
Chavier, Juan Aberdeen
Christopher, John Aberdeen
Church, Bill G. Sioux Falls
*Clark, B. S. Spearfish
Clark, C. J. Watertown
Cline, J. A. North Carolina
Collins, E. H. Gettysburg
Collins, James Hoven
Cornford, R. C. Rapid City
Cosand, M. R. Pierre
*Cottam, G. I. W. Sioux Falls
Cutshall, V. H. Sioux Falls
Cutshall, V. K. Sioux Falls
Daw, E. F. Sioux Falls
Dean, Roscoe Wess. Springs
Dean, Thomas Wess. Springs
DeClark, R. P. Sioux Falls
DeGeest, J. H. Miller
Delaney, R. J. Mitchell
Delaney, W. A., Jr. Mitchell
Dendinger, Wm. Vermillion
Desai, B. J. Watertown
Devick, J. S. Colton
Dewald, A. Rapid City
Dietrich, L. A. Bison
Donahoe, J. W. Sioux Falls
Driver, I. E. Aberdeen
Drummond, R. Rapid City
D'Souza, E. P. Aberdeen
*Dulaney, C. H. Ft. Meade
Dzintars, P. F. Rapid City
Ebbert, Larry Rapid City
Eckrich, J. A. Aberdeen
Eckrich, J. A., Jr. Aberdeen
*Eiringer, I. Sioux Falls
Elston, J. T. Rapid City
Ensberg, D. L. Sioux Falls
Entwistle, F. R. Sioux Falls
Epp, D. L. Freeman
Eulberg, M. Hot Springs

Fahrenwald, M. Conde
Farkas, E. C. Sioux Falls
Farrell, H. W. Sioux Falls
Fedt, Donald Watertown
Felker, J. Sioux Falls
Ferrell, M. R. Sioux Falls
Ferrell, R. Rapid City
Finley, R. C. Rapid City
Finney, L. Sioux Falls
*Fisk, R. G. Dell Rapids
Fletcher, H. Vermillion
Flora, G. Sioux Falls
Foley, R. J. Tyndall
Forshner, R. Hot Springs
*Fox, S. W. California
Freimark, L. Rapid City
Friefeld, S. Brookings
Friess, R. W. Sioux Falls
Fromm, H. E. Rapid City
Frost, D. M. Sioux Falls
Frost, H. L. Rapid City
Fuller, Wm. C. Sioux Falls
Gehring, S. Sioux Falls
Gerber, B. C. Aberdeen
Gere, R. G. Mitchell
Getz-Larsen, L. Sioux Falls
Giebink, R. R. Sioux Falls
Gilbert, F. J. Ft. Meade
Gillis, F. D. Mitchell
Golliher, W. N. Spearfish
Graham, D. Sioux Falls
Graven, S. Sioux Falls
Greenfield, D. L. Sioux Falls
Greenfield, R. E. Sioux Falls
Gregg, J. B. Sioux Falls
*Gregory, D. A. Milbank
Groote, C. Sioux Falls
Gross, H. Phil Sioux Falls
*Grove, M. S. Sioux Falls
Gryte, C. F. Huron
Guddal, W. N. Watertown
Gunnarson, R. E. Sioux Falls
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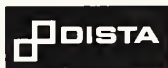
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Table of Contents: page 3

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300-mg.* Pulvules[®] and 600-mg.* Tablets



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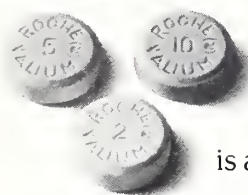
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A character all its own.



Valium (diazepam/Roche) is a benzodiazepine with a character all its own.

Pharmacologically, it is a potent skeletal muscle relaxant and anticonvulsant (in adjunctive use), as well as an antianxiety agent. Pharmacokinetically, only Valium provides active *diazepam* as well as the active metabolites 3-hydroxydiazepam, desmethyldiazepam and oxazepam.

But the individual character of Valium is even more apparent clinically than pharmacokinetically. And far more significant. That's because of the patient response obtained with Valium. A response which brings a calmer frame of mind. A response which has a pronounced effect on the somatic symptoms of anxiety, particularly muscular tension. A response which helps the patient feel more like himself again because of the way Valium reduces the overwhelming symptoms of anxiety and psychic tension.

Another important aspect of the clinical character of Valium is safety. Though drowsiness, ataxia and fatigue are possible, these and more serious side effects are rarely a problem. Of course, as with all CNS-acting drugs, patients taking Valium should be cautioned against driving, operating dangerous machinery or the simultaneous ingestion of alcohol.

Unquestionably, many psychotherapeutic agents, including other benzodiazepines, have antianxiety effects. But one fact remains: you get a certain kind of patient response with Valium. It's a response you want. A response you know. A response you trust as part of your overall management of anxiety and psychic tension.

Valium®^{IV}
diazepam/Roche
2-mg, 5-mg, 10-mg scored tablets
a prudent choice in psychic
tension and anxiety

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.



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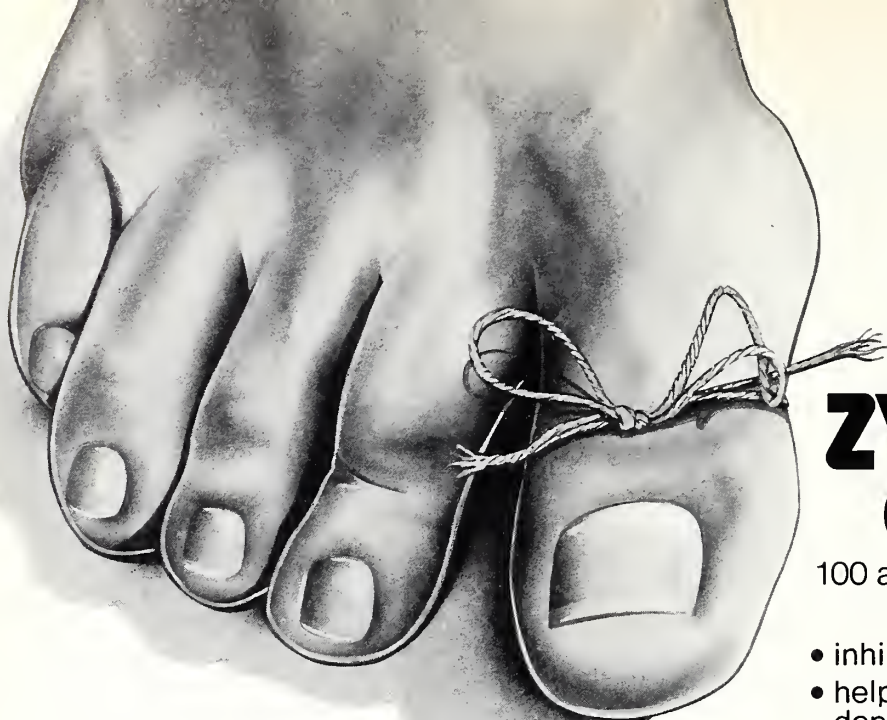
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SCIENTIFIC ARTICLES

- 5** Major/Multiple Congenital Anomalies and
Intrapartum Fetal Heart Rate Patterns
Shailaja M. Didolkar, M.D., FACOG
Milton Mutch, M.D., FACOG
- 27** Clinicopathological Conference
Two Year Old Child with Pain on Urination
J. H. Hoskins, M.D.
L. G. Thatcher, M.D.
D. G. Nordstrom, M.D.
J. F. Barlow, M.D.

FEATURES

- 15** President's Page
- 17** South Dakota AFP Chapter News
- 22** Laboratory Aids
South Dakota Pathology Page
- 34** Future Meetings



A reminder

ZYLOPRIM[®]

(allopurinol)

100 and 300 mg scored Tablets

- inhibits uric acid formation
- helps prevent urate crystal depositions in synovia
- reduces risk of uric acid lithiasis

INDICATIONS AND USE: This is not an innocuous drug and strict attention should be given to the indications for its use. Pending further investigation, its use in other hyperuricemic states is not indicated at this time.

Zyloprim[®] (allopurinol) is intended for:

1. treatment of gout, either primary, or secondary to the hyperuricemia associated with blood dyscrasias and their therapy;
2. treatment of primary or secondary uric acid nephropathy, with or without accompanying symptoms of gout;
3. treatment of patients with recurrent uric acid stone formation;
4. prophylactic treatment to prevent tissue urate deposition, renal calculi, or uric acid nephropathy in patients with leukemias, lymphomas and malignancies who are receiving cancer chemotherapy with its resultant elevating effect on serum uric acid levels.

CONTRAINDICATIONS: Use in children with the exception of those with hyperuricemia secondary to malignancy. The drug should not be employed in nursing mothers.

Patients who have developed a severe reaction to Zyloprim should not be restarted on the drug.

WARNINGS: ZYLOPRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION. In some instances a skin rash may be followed by more severe hypersensitivity reactions such as exfoliative, urticarial and purpuric lesions as well as Stevens-Johnson syndrome (erythema multiforme) and very rarely a generalized vasculitis which may lead to irreversible hepatotoxicity and death.

A few cases of reversible clinical hepatotoxicity have been noted and in some patients asymptomatic rises in serum alkaline phosphatase or serum transaminase have been observed. Accordingly, periodic liver function tests should be performed during the early stages of therapy, particularly in patients with pre-existing liver disease. Patients should be alerted to the need for due precautions when engaging in activities where alertness is mandatory.

Nevertheless, iron salts should not be given simultaneously with Zyloprim. This drug should not be administered to immediate relatives of patients with idiopathic hemochromatosis.

In patients receiving Purlinethol[®] (mercaptopyrine) or Imuran[®] (azathioprine), the concomitant administration of 300-600 mg of Zyloprim per day will require a reduction in dose to approximately one-third to one-fourth of the usual dose of mercaptopyrine or azathioprine. Subsequent adjustment of doses of Purlinethol or Imuran should be made on the basis of therapeutic response and any toxic effects.

Usage in Pregnancy and Women of Childbearing Age. Zyloprim[®] (allopurinol) should be used in pregnant women or women of childbearing age only if the potential benefits to the patient are weighed against the possible risk to the fetus.

PRECAUTIONS: Some investigators have reported an increase in acute attacks of gout during the early stages of allopurinol administration, even when normal or sub-normal serum uric acid levels have been attained.

It has been reported that allopurinol prolongs the half-life of the anticoagulant, dicumarol. This interaction should be kept in mind when allopurinol is given to patients already on anticoagulant therapy, and the coagulation time should be reassessed.

A fluid intake sufficient to yield a daily urinary output of at least 2 liters and the maintenance of a neutral or, preferably, slightly alkaline urine are desirable to (1) avoid the theoretic possibility of formation of xanthine calculi under the influence of Zyloprim therapy and (2) help prevent renal precipitation of urates in patients receiving concomitant uricosuric agents.

Patients with impaired renal function require less drug and should be carefully observed during the early stages of Zyloprim administration and the drug withdrawn if increased abnormalities in renal function appear.

In patients with severely impaired renal function, or decreased urate clearance, the half-life of oxipurinol in the plasma is greatly prolonged. Therefore, a dose of 100 mg per day or 300 mg twice a week, or perhaps less, may be sufficient to maintain adequate xanthine oxidase inhibition to reduce serum urate levels. Such patients should be treated with the lowest effective dose, in order to minimize side effects.

Mild reticulocytosis has appeared in some patients.

As with all new agents, periodic determination of liver and kidney function and complete blood counts should be performed especially during the first few months of therapy.

ADVERSE REACTIONS:

Dermatologic: Because in some instances skin rash has been followed by severe hypersensitivity reactions, it is recommended that therapy be discontinued at the first sign of rash or other adverse reaction (see WARNINGS). Skin rash, usually maculopapular, is the adverse reaction most commonly reported.

Exfoliative, urticarial and purpuric lesions, Stevens-Johnson syndrome (erythema multiforme) and toxic epidermal necrolysis have also been reported.

A few cases of alopecia with and without accompanying dermatitis have been reported.

In some patients with a rash, restarting Zyloprim (allopurinol) therapy at lower doses has been accomplished without untoward incident.

Gastrointestinal: Nausea, vomiting, diarrhea, and intermittent abdominal pain have been reported.

Vascular: There have been rare instances of a generalized hypersensitivity vasculitis or necrotizing angitis which have led to irreversible hepatotoxicity and death.

Hematopoietic: Agranulocytosis, anemia, aplastic anemia, bone marrow depression, leukopenia, pancytopenia and thrombocytopenia have been reported in patients, most of whom received concomitant drugs with potential for causing these reactions. Zyloprim[®] (allopurinol) has been neither implicated nor excluded as a cause of these reactions.

Neurologic: There have been a few reports of peripheral neuritis occurring while patients were taking Zyloprim. Drowsiness has also been reported in a few patients.

Ophthalmic: There have been a few reports of cataracts found in patients receiving Zyloprim. It is not known if the cataracts predated the Zyloprim therapy. "Toxic" cataracts were reported in one patient who also received an anti-inflammatory agent; again, the time of onset is unknown. In a group of patients followed by Gutman and Yu for up to five years on Zyloprim therapy, no evidence of ophthalmologic effect attributable to Zyloprim was reported.

Drug Idiosyncrasy: Symptoms suggestive of drug idiosyncrasy have been reported in a few patients. This was characterized by fever, chills, leukopenia or leukocytosis, eosinophilia, arthralgias, skin rash, pruritus, nausea and vomiting.

OVERDOSAGE: Massive overdosing, or acute poisoning, by Zyloprim has not been reported.

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Major/Multiple Congenital Anomalies and Intrapartum Fetal Heart Rate Patterns

Shailaja M. Didolkar, MD, FACOG*

Milton Mutch, MD, FACOG**

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University of South Dakota School of Medicine
and the South Dakota Perinatal Project

ABSTRACT

Intrapartum fetal heart rate (FHR) patterns of nine mothers who delivered congenitally malformed fetuses were reviewed to see whether it could alert the obstetrician about the fetal anomalies and prevent C-section for grossly anomalous fetus. Bizarre fetal heart rate patterns are common in fetuses with CNS anomaly. Fixed heart rate and/or late decelerations and terminal bradycardias was a consistent finding in fetuses with severe CNS anomalies.

Many a time an obstetrician faces a problem of doing a C-section for severe fetal distress and finds out to his dismay that the baby is severely malformed and is incompatible for extrauterine survival. In such situations, C-section could have been avoided if fetal anomalies were diagnosed. The purpose of this study is to see whether intrapartum fetal heart rate pattern could indicate major/multiple congenital anomalies incompatible for fetal survival. We have reviewed all major congenital malformed live born at Sioux Valley Hospital during the year 1977. One case of Trisomy 13 is added to these cases which prompted us to look into this problem.

Anomalies not involving CNS did not present as a fetal distress unless associated with prenatal or intrapartum reasons to explain it. Five out of nine mothers (55.5%) were delivered by C-section. Fetal distress was the indication in four of them. Because six out of nine mothers presented with polyhydramnios, SGA, or breech presentation, this review reemphasizes the need for careful evaluation of fetal anomalies in these prenatal complications.

MATERIALS AND METHOD

At Sioux Valley Hospital from January 1, 1977, to December 31, 1977, a total of 1,405 patients were delivered. Eight major/multiple malformed babies were live born. This total number of malformed fetuses included those diagnosed at delivery and the first four days of neonatal period. In all except one neonatal death, the diagnosis of congenital anomalies was confirmed by autopsy, while in live babies, it was confirmed by an appropriate laboratory investigation. One case of Trisomy 13 is added to the series and findings were confirmed by autopsy and karyotype.

Table I shows briefly their prenatal history, intrapartum fetal heart rate (FHR) pattern and neonatal course. External and internal fetal heart rate monitoring was done; intrauterine pressure catheters were not used. Abnormal fetal heart rate patterns are shown in Figures 1 through 6. FHR patterns are diagnosed according to Hon's classification.¹

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**1200 South Euclid Avenue, Sioux Falls, S.D. 57105.

TABLE 1
Clinical summary of 9 patients' prenatal, intrapartum, and neonatal record
FHR Monitoring Delivery

Case	Age (yrs)	Gr-Para	Wks Gest	Prenatal Complications	Int/Ext Monitor	FHR Pattern	Method of Del.	Indication	Birth Wt. (gms)	Apgar 1&5 min	Neonatal Course Anomalies	Alive/Dead (hrs)
1	26	Primip	40	Preeclampsia, mild. Resolved by bed rest.	Int	No B/B var., late dec. (undulating pattern) Baseline heart rate 60, bradycardia. (Fig 1 & 2)	C-sect	Fetal distress	3515 (AGA)	1/0	Severe int. hydrocephalus, atresia of aqueduct of Sylvius. Meningo-myelocele, T4-5 lumbar spina bifida. PDA	D
2	24	Primip	35	Mild spotting, 2nd trimester	Ext	Record poor, very short period	C-sect	Primigravida breech & prolonged rupture of membranes	2665 (AGA)	8/9	Sacral meningo-myelocele, L5-3, hydrocephalus, neurogenic bladder, rectal incontinence.	A
3	26	g2p0010 (ETP)	Term 40+	None. E ₃ at 36 wks normal. Nonstress test 2 days prior to labor normal.		Flat baseline, late dec. pH 7.06 (Fig 3 & 4)	C-sect	Fetal distress	3062	2/5	Trisomy 13	D (3)
4	19	Primip	42	None. Dx of anencephaly prior to labor	Not monitored		Vaginal		2551 (SGA)	5/5	Anencephaly	D (48)
5	24	g1p1001	30-32	None	Not monitored. Admitted with prolapsed cord		C-sect	Prolapsed cord.	1559 (AGA)	1/3	Absent abd. wall (Prune belly synd.), bil. club feet, polycystic renal disease	D same day
6	30	g3p2002	40	None	Ext	Normal	Vaginal		2750 (AGA)	7/9	Hypoplastic left heart syndrome	D (26)
7	25	g3p2002	39	None	Ext	Normal	Vaginal (very rapid)		3147 (AGA)	3/4	Pierre-Robin syndrome	A/W
8	24	g2p1000	727-28	Polyhydramnios referred in labor	Ext	Normal	Vaginal		2098	4/3	?Klippel-Feil syndrome, web neck, bil. club feet, anasarca.	D (72)
9	26	g2p1100	37	1. Preeclampsia superimposed on essential hypertension. 2. Polyhydramnios. 3. Meds—Valium, Aldactazide, C-Ron tid	Int	Reduced B/B var., prolonged severe bradycardia with tetanic contractions. (Fig 5 & 6)	C-sect	Fetal distress	1786 (SGA)	8/9	Esophageal atresia	A

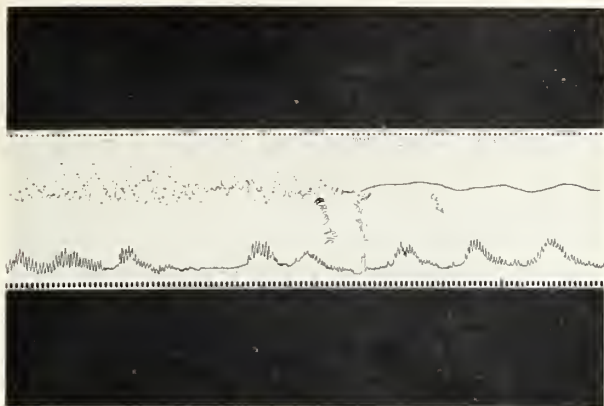


Figure 1

FHR-UC pattern of a fetus with severe internal hydrocephalus with lumbar meningocele (Case No. 1). Poor external monitor record. Internal monitor shows absolute lack of B/B variability. Initial baseline heart rate 120-130/min. Late deceleration, appears more to be as undulating heart rate.

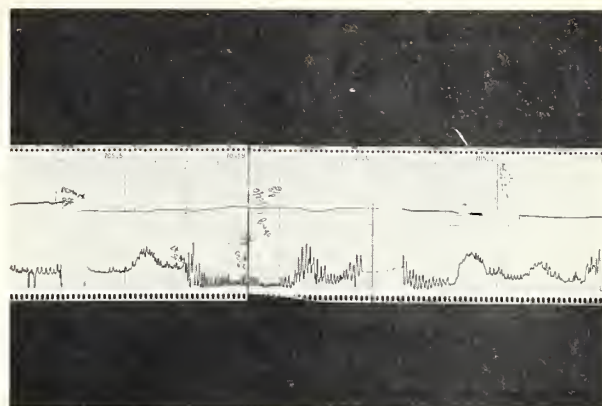


Figure 2

Continued tracing from Fig. 1. Notice decreased baseline heart rate to 90/min. Shortly after the end of tracing, patient was delivered by C-section with apgar 1/0 at 1 and 5 minutes.

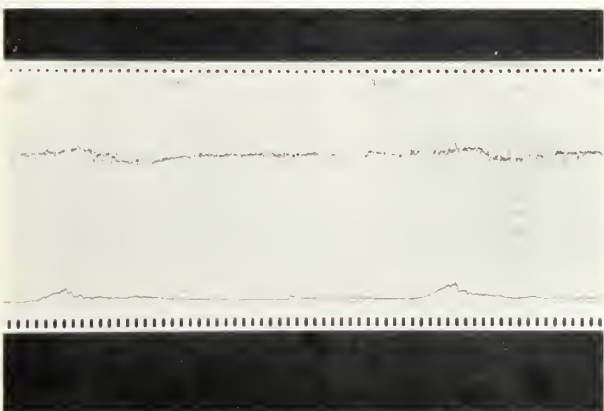


Figure 3

External monitor record of fetus with Trisomy 13 (Case No. 3). Mother in early labor, persistent late decelerations.



Figure 4

Same patient with internal monitor. Absolute lack of B/B variability and persistent late decelerations. Fetal scalp sample pH 7.06.

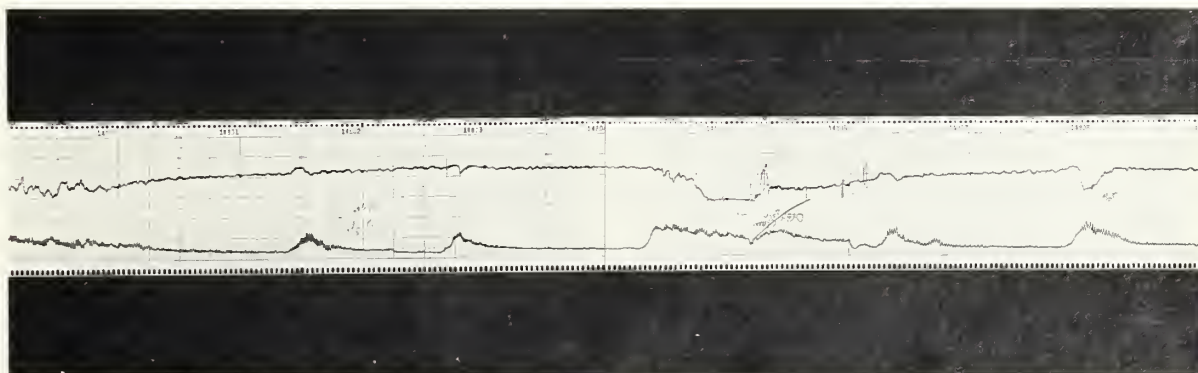


Figure 5

FHR-UC tracing at 3 different times (Case No. 8) during elective induction showing two severe and prolonged episodes of bradycardia with prolonged and tetanic contractions. Notice B/B variability in between contractions.

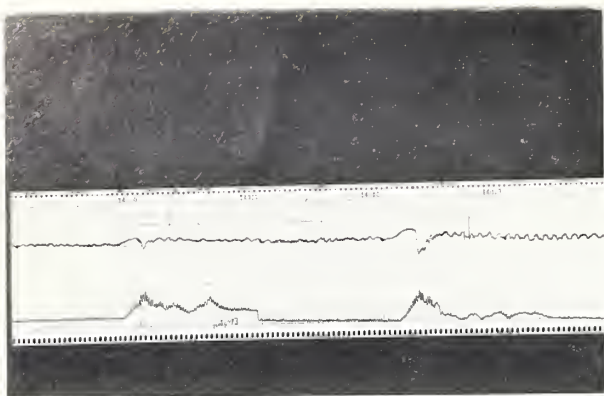


Figure 6

Continued record from Fig. 5. Normal duration uterine contractions, mild variable decelerations. Shortly after the end of the tracing, patient was delivered by C-section for fetal distress with apgar scores 8/9 at 1 and 5 minutes.

RESULTS

Out of nine cases, seven were term pregnancies and two were preterm. Two mothers had polyhydramnios. Of the two mothers who had preeclampsia, one had severe superimposed preeclampsia on essential hypertension and the other had mild preeclampsia which resolved with bed rest. Five mothers were delivered by C-section; four were for fetal distress and one for breech presentation in a primigravida with prolonged premature rupture of membranes.

Seven neonates were term and two were preterm. Out of seven neonates, two were small for gestational age (SGA) which were diagnosed after birth. Six out of nine infants had apgar scores equal to or less than five at five minutes. Six neonates died either immediately or within the first four days of birth. Four babies had central nervous system (CNS) anomalies which included the Trisomy 13 baby. Five had other rare anomalies.

Intrapartum fetal heart rate pattern of two out of four fetuses with CNS anomalies showed absolute lack of beat to beat variability (fixed heart rate) and late decelerations. The remaining fetus with anencephaly was not monitored because she was diagnosed prenatally. Three of these four patients were delivered by C-section; two for fetal distress and one for breech presentation in primigravida with prolonged rupture of membranes.

Of the five infants with other rare anomalies, three had normal fetal heart rate patterns. One FHR was not recorded as mother was admitted with prolapsed umbilical cord and breech presentation. The remaining fetus showed good beat to beat variability with repeated prolonged episodes of

bradycardia with decreasing beat to beat variability during decelerations. These decelerations were associated with tetanic prolonged uterine contractions. With normal contractions, decelerations were mild to moderate variable. At the end of the tracing, there was a sinusoidal pattern (Figures 5 & 6). Out of these five patients, three were delivered vaginally and two were delivered by C-section, one for prolapsed umbilical cord and the other for fetal distress.

DISCUSSION

According to the presence or absence of CNS involvement in our cases, fetal anomalies were divided in two major categories. FHR pattern in fetuses with CNS anomalies was consistently abnormal. Characteristically it showed an absolute lack of beat to beat variability and late decelerations (Figures 1, 2, 3, & 4). These FHR abnormalities could not be explained by other causes. Since the CNS controls the fetal heart rate,^{2,3} anomalies involving the CNS could result in an abnormal fetal heart rate pattern.

Similar abnormal FHR pattern has been described in anencephalic fetuses by Hon and Lee⁴ and Calderyo-Barcia, et al.² Unfortunately FHR was not recorded in the anencephalic fetus in our case because anencephaly was diagnosed prenatally.

FHR of Trisomy 13 fetus showed similar characteristic abnormal patterns with absolute lack of beat to beat variability and late decelerations. Similar findings of late decelerations have been reported in Trisomy 18 and 21.^{5,6,7} It was interesting to note that their prepartum FHR patterns corresponded well to our intrapartum FHR pattern.

All infants without CNS anomalies had normal FHR pattern except one who was SGA and mother had a severe superimposed preeclampsia on essential hypertension. Beat to beat variability of FHR was average. Decelerations were mainly related to prolonged tetanic uterine contractions. With normal uterine contractions, they were mild to moderate variable decelerations and at birth apgar score was 8/9. Neonatal course was normal except for feeding problems due to esophageal atresia. This would indicate that etiology of fetal distress was not related to fetal anomaly.

Bizarre fetal heart rate patterns or fixed heart rate and/or late decelerations were consistent findings in cases with CNS anomalies. Hence, unexplained fetal distress in labor presenting as fixed heart rate and/or late decelerations should alert the physician about severe CNS anomalies. Ultrasound or x-ray examination will help to conform these anomalies. These patterns are very similar to

agonal patterns described in the literature.⁸⁻¹⁴ Indeed, in our cases the fetuses showing similar patterns died within first four days of life. The question is were these patterns indicative of CNS anomaly or severe compromised fetal intrauterine environment or both? Anomalies without CNS involvement in general will not present as fetal distress unless they were associated with prenatal or intrapartum complications to explain this. It is noteworthy that hypoplastic left heart syndrome in one fetus showed completely normal FHR pattern.

Since six out of nine mothers presented with prenatal complications, namely polyhydramnios, SGA infant, prematurity and malpresentations, especially breech presentation, this review reemphasizes the need for careful evaluation for fetal anomalies in the above prenatal complications.¹⁵⁻¹⁷

SUMMARY

Intrapartum fetal heart rate (FHR) patterns of nine mothers who delivered congenitally malformed fetuses were reviewed to see whether it could alert the obstetrician about the fetal anomalies and prevent C-section for grossly anomalous fetus. The fetuses were divided mainly in two groups according to CNS involvement. Fixed heart rate and/or late decelerations or prolonged bradycardias was a consistent finding in fetuses with severe CNS anomalies. Anomalies not involving CNS did not present as a fetal distress unless associated with prenatal or intrapartum reasons to explain it. Because six out of nine mothers presented with polyhydramnios, SGA, prematurity, or breech presentation, this review reemphasizes the need for careful evaluation of fetal anomalies in these prenatal complications.

Special acknowledgement to Deborah Jones for her help in preparing this manuscript.

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continued from page 34

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S **D** *President's Page*

The Federal Trade Commission, in 1975, without any prior notice or investigation, issued a complaint challenging our ethical principles applicable to advertising and solicitation by physicians. A revered principle of the American Medical Association, of local Societies, and adhered to by all ethical physicians frowning upon crass advertising was negated completely by a bureaucratic federal commission's edict without any reason other than medicine was a **business**.

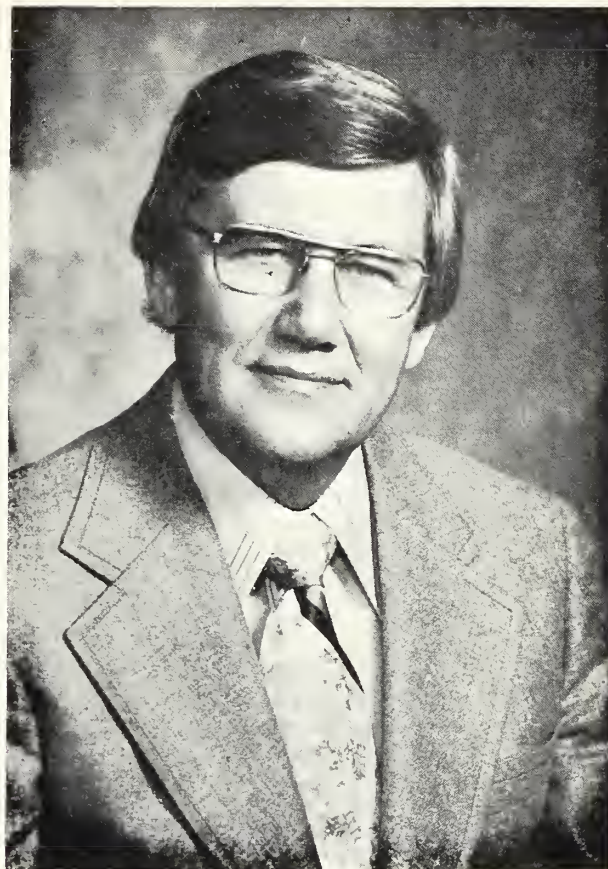
Yes, as men and women trained in a noble and learned profession, you are now on notice that parts of our government claim that the medical profession is not a profession but a business—and that health care is now the FTC's business!

Consequently, the medical profession has bowed to this "wise and sage" edict, but has requested that local and county medical societies be allowed to set the standards of ethical promotional practices. After all, who is better equipped and more knowledgeable in determining factual ethical advertising than we at the local level?

We have then contended that professional associations, including the AMA, have not only the right but the responsibility and duty to see that advertising by their members be limited to truthful, objective and verifiable information that will help to enable patients to make an informed choice among physicians.

But the staff of the Federal Trade Commission flatly rejected this position, and they have insisted that advertising by physicians should be regulated exclusively by the government. This is essentially reprehensible since it implies that professional men and women cannot be trusted to regulate themselves in the public interest. It boggles the mind when a federal agency directs a select group of people, the physicians, to advertise their wares (something they have never done or deemed necessary) and then sets up their own set of rules as to how and what they can advertise!

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And so the AMA again fought this battle in court before an administrative law judge—(Guess who? —A former prosecutor for the FTC), who decreed that the AMA was organized for profit since it had offered its members such things as retirement plans and had opposed certain forms of national health insurance. This administrative law judge's order demands that the AMA may not establish ethical guidelines governing advertising and solicitation of its members unless first obtaining the "permission and approval of the Federal Trade Commission".

The AMA will continue to fight this battle against "Big Brother" which would, on the surface, seem to be an unconscionable affront to the ethics of all good physicians. Eighty five percent of the physicians who belong to the South Dakota State Medical Association belong to the AMA. It should be 100%. After all, "United we stand, divided—we end up nationalized".

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D. B. Reaney, M.D., President
South Dakota State Medical Association

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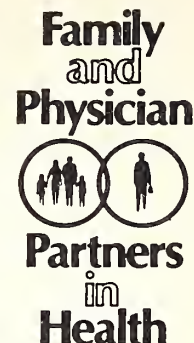
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South Dakota Academy of Family Physicians Awards Memorial Merit Scholarships



Pictured above (l to r) are Memorial Merit Scholarship recipients John Jones, M.D. and Craig K. Hansen, M.D. being given their certificates and checks by SDAFP President Burton O. Lindbloom.

Two 1979 USDSM graduates, John Jones, M.D. and Craig K. Hansen, M.D., were awarded the SDAFP Memorial Merit Scholarship on June 27, 1979. The awards were given at an informal ceremony at the Family Practice Center in Sioux Falls, the presentations being made by SDAFP President Burton O. Lindbloom of Pierre.

This award is presented to a USDSM graduate who has been accepted into a family practice residency, beginning the year of his graduation. This award, a \$1,000 cash scholarship, is based upon need and possibly upon scholarship, but not necessarily upon scholarship. A special SDAFP Committee has developed the criteria and carries out the application and review process.

Both Drs. Jones and Hansen have begun their three year family practice residency in the Sioux Falls Family Practice Residency program. Both

physicians are South Dakota natives and plan to practice family medicine in the state of South Dakota upon completion of the residency program.

SDAFP Memorial Lecture

SDAFP Board of Directors, has sanctioned the "SDAFP Memorial Lecture", to be given at each of the chapter sponsored Black Hills Seminars.

This lecture is to be given by an active, practice affiliate or resident affiliate member of SDAFP on a topic of the speaker's preference. Suggested topics are available from the Education Committee, based upon the cyclic core of knowledge for family practice.

Member applications for the privilege of being selected for this lecture must be available to the state office by April 1 of each year for the Summer Seminar and October 15 for the Winter Seminar. The application will be a letter of intent to be selected and an outline, with references, of the proposed lecture, including the title. The speaker selected for each of these lectures will be handled by the SDAFP Education Committee through a review process.

This memorial lecture, dedicated to former SDAFP members now deceased, will carry the honorarium award of \$200. Your participation is invited.

New FP Residency Director Named

Robert Cloar, M.D., of Scottsdale, AZ, has been named the new Director of the Sioux Falls Family Practice Residency. Dr. Cloar, a native of Kentucky, has been in practice for 15 years, and has been a member of the part-time faculty of the Scottsdale Family Practice Program while in practice.

Dr. Cloar will begin his duties in Sioux Falls October 1, 1979.

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helps control abnormal motor activity
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In this double-blind study, twenty patients having G.I. series and exhibiting spasm were randomly selected to receive either 2 cc. of Bentyl or sodium chloride intramuscularly. Ten minutes after the injection another radiograph was taken . . .

. . . Bentyl produced definite relaxation in 8 of 10 patients. The sodium chloride produced relaxation in only 3 of 10. No side effects occurred in either group of patients.



Pylorospasm has almost totally blocked passage of barium meal.



Barium meal beginning to pass 10 minutes after intramuscular injection of 20 mg. Bentyl.

"The correlation of spasm relief and drug given was excellent."

*This drug has been classified "probably" effective in treating functional bowel/irritable bowel syndrome.

†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:

King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

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Brief Summary

INDICATIONS

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the following indications as "probably" effective:

For the treatment of functional bowel/irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

THESE FUNCTIONAL DISORDERS ARE OFTEN RELIEVED BY VARYING COMBINATIONS OF SEDATIVE, REASSURANCE, PHYSICIAN INTEREST, AMELIORATION OF ENVIRONMENTAL FACTORS.

For use in the treatment of infant colic (syrup).

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloroduodenal stenosis); paralytic ileus, intestinal atony of the elderly or debilitated patient, unstable cardiovascular status in acute hemorrhage; severe ulcerative colitis; toxic megacolon complicating ulcerative colitis; myasthenia gravis. **WARNINGS:** In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. **PRECAUTIONS:** Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy. Use with caution in patients with: Autonomic neuropathy, Hepatic or renal disease, Ulcerative colitis. Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon. Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension. Hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.

Do not rely on the use of the drug in the presence of complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. **ADVERSE REACTIONS:** Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia; urinary hesitancy and retention; blurred vision and tachycardia; palpitations; mydriasis; cycloplegia; increased ocular tension; loss of taste; headache; nervousness; drowsiness; weakness; dizziness; insomnia; nausea; vomiting; impotence; suppression of lactation; constipation; bloated feeling; severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating. With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. **DOSE AND ADMINISTRATION:** Dosage must be adjusted to individual patient's needs.

Usual Dosage: Bentyl 10 mg. capsule and syrup: *Adults:* 1 or 2 capsules or teaspoonfuls syrup three or four times daily. *Children:* 1 capsule or teaspoonful syrup three or four times daily. *Infants:* ½ teaspoonful syrup three or four times daily. (May be diluted with equal volume of water.) Bentyl 20 mg.: *Adults:* 1 tablet three or four times daily. Bentyl Injection: *Adults:* 2 ml. (20 mg.) every four to six hours intramuscularly only. **NOT FOR INTRAVENOUS USE. MANAGEMENT OF OVERDOSE:** The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal. Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested. If indicated, parenteral cholinergic agents such as Urecholine[®] (bethanechol chloride USP) should be used.

Product Information as of October, 1978.

Injectable dosage forms manufactured by CONNAUGHT LABORATORIES, INC., Swiftwater, Pennsylvania 18370 or TAYLOR PHARMACAL COMPANY, Decatur, Illinois 62525 for MERRELL-NATIONAL LABORATORIES, Division of Richardson-Merrell Inc., Cincinnati, Ohio 45215, U.S.A.

S D Laboratory Aids

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Sputum Cultures

Determination of the etiologic agent of lower respiratory infections remains a problem. Even excluding special methods of cultivation required by mycobacterial, fungal, viral and newly recognized agents such as mycoplasma pneumoniae which can cause primary atypical pneumonia and legionella pneumophila which can cause Legionaire's disease, the proper identification of the garden variety pathogens such as pneumococcus, staphylococcus, streptococcus, gram negative enteric bacilli and haemophilus present considerable problems.

The reasons for the difficulty are interrelated and centered about proper collection of the specimen. Material from the lower respiratory tract is hard to obtain especially in sick patients with a poor cough mechanism and in children. The situation is compounded when one appreciates that many of the pathogens which can cause lower respiratory tract infections reside transiently or permanently in the oral-pharyngeal region. Hence, material cultured from this region will yield potential pathogens which may or may not be culprit of the pneumonic process.

The collection of the specimen can be facilitated by making certain that only material obtained from a deep cough is submitted. A single specimen obtained preferably early in the morning is most suitable. To procure such a specimen requires effort and instruction to the patient on the part of the nursing service and physicians. If a deep cough specimen cannot be obtained, the respiratory therapist who is available in an increasing number of hospitals can be of aid in obtaining the sputum sample.

Once the material is obtained, a method for determining its suitability is necessary. **Gram stain** can be performed to determine—1) the number of squamous epithelial cells (SEC) per low power field, 2) the number of polymorphonuclear leukocytes (PMN) per low power field, 3) whether there is a predominant type of organism present e.g. gram positive diplococci. Criteria differ but we feel that less than 25 SEC, greater than 25 PMN per low power field, and a predominance of an organism particularly if associated with PMN are indicators of an adequate sputum sample. If these criteria are not met, the physician should be notified. Another specimen should be obtained if possible before culture is performed since misleading information can arise from culturing inappropriate samples.

John F. Barlow, M.D.
Pathologist

Merrell

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Cincinnati, Ohio 45215, U.S.A.

Two Year Old Child with Pain on Urination

J. H. Hoskins, M.D.*

L. G. Thatcher, M.D.**

D. G. Nordstrom, M.D.***

Discussers

John F. Barlow, M.D.****

Pathologist Editor

Case No. 719-225

This 2-year-old caucasian male was admitted to Sioux Valley Hospital for evaluation of pain on urination.

The patient was seen ten days prior to admission because of the above complaint. Urinalysis showed pyuria but no hematuria but the symptoms did not abate after five days of sulfasoxazole. A culture, which showed no growth in 24 hours, was taken and the patient was started on nalidixic acid before the culture returned. The patient continued to have dysuria and apparently some decreased stream on voiding as well as dribbling. An excretory urogram revealed possible clots in the bladder. An erythrocyte sedimentation rate and an antistreptolysin O titer were within normal limits. The blood urea nitrogen and creatinine were within normal limits. He was referred for further evaluation.

There was no history of serious illnesses or prior hospitalizations. Review of systems was unremarkable. The patient's growth and development had been excellent and he had five siblings in good health although one sibling had diabetes.

PHYSICAL EXAMINATION: pulse 120/min. and regular, blood pressure 100 systolic and 60 diastolic, respirations 28/min. and regular, temperature 98.6°F. The child was quite irritable but had no other abnormalities. Examination of the head and neck was unremarkable. The chest was clear to auscultation and percussion. The heart showed a normal heart size with a regular rhythm and no murmurs. The abdomen was soft with no palpable tenderness, organs, or masses. Neurologic examination was within normal limits. A 1 cm right inguinal lymph node and 2-3 slightly enlarged left inguinal lymph nodes were observed.

At the time of cystoscopy a rectal examination was carried out and the prostate was felt to be enlarged but normal in consistency. There were 200 cc's of residual urine in the bladder. On cystoscopy the distal urethra was unremarkable. The prostate seemed enlarged with polypoid frond-like projections which extended to the floor and roof of the bladder. No other lesion was noted in the bladder. The ureteral orifices were unremarkable. A cystourethrogram showed a lesion in the posterior urethra. Intravenous pyelogram showed a normal upper collecting system. A chest film and bone survey series by x-ray were unremarkable.

LABORATORY DATA: Urinalysis—yellow, slightly turbid, specific gravity 1.026, pH 6.0, negative for protein, glucose, ketone bodies, bile, large amount of hemoglobin; sediment 1-3 wbc/hpf, 3-5 rbc/hpf no casts. Hemoglobin 12.4 gms/dl, hematocrit 35 vol/dl, red count 4.85 million/mm³, mean corpuscular, hemoglobin 26 micrograms, mean corpuscular volume 75 cubic micra, mean corpuscular, hemoglobin concentration 35%, total leukocyte count 8,200/mm³ with 26% segmented neutrophils, 3% neutrophilic bands, 1% eosinophils, 1% basophils and 65% normal lymphocytes. The red cells were normochromic and normocytic and the platelets normal in number and morphology. A urine culture showed no growth in 24

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hours. A bone scan was negative. A creatinine was 0.7 mgs/dl. A bone marrow examination showed no diagnostic abnormality. Diagnosis was made by histologic examination of a bladder biopsy.

DR. HOSKINS: I would like to point out that the referring physician was a family practitioner, Dr. Richard A. Jongewaard, who trained in the Sioux Falls program and investigated this patient very well before he arrived. The patient had had a urinalysis and an intravenous pyelography so that the abnormal urinary tract findings had already been demonstrated. All that was needed was for us to establish a pathologic diagnosis. The cystoscopy was very interesting in this patient. Usually children have a prominent verumontanum and a small prostate. In this patient the prostate was markedly enlarged, particularly toward the bladder neck on the right side and near the floor of the bladder. The examination was very similar to an elderly man with prostatic hyperplasia. There were grapelike or papillary fronds which hung down from the roof of the bladder and involved the floor of the bladder and probably produced a ball valve like obstruction of the bladder neck opening. Some of the papillary fronds seemed to extend from the prostate into the bladder. These were resected and sent for both frozen section and permanent section. The remainder of the bladder showed no significant abnormalities. The ureteral orifices seemed quite close to the bladder neck but were not obstructed. Cystogram of the bladder showed multiple filling defects in and around the base of the bladder and prostate. (Fig. 1)



Figure 1

Cystourethrogram with polypoid filling defects at bladder outlet.

The frozen section and final pathologic report revealed an embryonal rhabdomyosarcoma. The case history is typical of that entity. The x-ray picture is characteristic but these children often have a palpable bladder or palpable suprapubic mass which was

not present in this case. The most common presenting symptom of this tumor is obstruction. Hematuria may occur due to the infiltrating submucosal tumor which may develop secondary ulceration. Secondary bladder infection may also occur.

After the diagnosis was made, I tried to review the literature since I had limited experience with this lesion. Indeed, the entity is quite rare. At the University of Iowa between 1933 and 1973 only one case of rhabdomyosarcoma of the prostate was recorded. The University of Michigan had a total of 14 cases over many years experience.¹² Williams in England¹³ reported on 24 cases over a long period. No single experience was very large. In a review article in *Cancer* in 1972,⁸ 124 cases were reviewed from the literature and 14 cases were added. In that review, 23 cases before 1967 had a 14% survival rate at 5 years. Up to that time, the only approach had been radical surgical extirpation of the prostate, seminal vesicles and bladder. Thirty-eight cases followed since 1967 showed a 71% survival. In the latter series the higher survival rate was obtained by the use of chemotherapy and radiation as well as less extensive surgery.

In general, the prognosis for embryonal rhabdomyosarcomas of prostate origin are worse than for those from bladder origin. However, seeing as many of the lesions arise in the region of the trigone, it may be difficult to determine the exact site of origin. There have been occasional reports of small localized rhabdomyosarcomas of the bladder which have been removed by local excision with adequate margins. Although the number of cases is small, the general impression is that radical surgical removal leads to many complications and probably is not going to be the preferred therapy of the future. Debulking of the tumor followed by chemotherapy and/or radiation is probably productive of better results in both survival and future quality of life.

At surgery there was a marked area of demarcation from what appeared to be normal bladder mucosa and infiltrating tumor arising from the prostate. A radical retropubic prostatectomy was performed with removal of the prostate, seminal vesicles, and portions of the base of the bladder. The prostate was quite adherent to the rectum. In spite of the good demarcation of tumor clinically, microscopically there was tumor extending to the margins of resection. Radical cystectomy and removal of the rectum were considered at the time of surgery, but debulking of the tumor was felt to be the better choice.

DR. BARLOW: Tissue removed from the junction of the bladder neck, border of the right ureter and

prostate showed extensive embryonal rhabdomyosarcoma. This slide shows one of the polypoid masses described clinically. (Fig. 2) You will note that there is more cellularity just beneath the region from which the surface epithelium has been eroded. This increased cellularity is referred to as the cambium layer and is characteristic of these tumors. On higher power, large strap or tandem cells with multiple nuclei and long streaming eosinophilic cytoplasm can be seen. (Fig. 3) An even higher power shows cross striations in the strap cells (Fig. 4). Embryonal rhabdomyosarcoma is the most common soft tissue tumor of childhood. When the tumor projects from a body orifice or into a hollow viscus such as the bladder, a grapelike or botryoid appearance occurs. This has been described in the vagina and has been referred to as sarcoma botryoides. However, the lesion is an embryonal rhabdomyosarcoma. Roughly, 35 to 50% of embryonal rhabdomyosarcomas arise in the head and neck, 30% in the extremities and trunk, and 20 to 30% in the genitourinary tract. Tumors of the orbit, nose, nasopharynx, pharynx, ear, or genitoruinary tract are most common in the years right after birth. There is a second peak of these tumors in the paratesticular structures in teenagers. The genitourinary tract tumors usually involve the bladder, prostate, or vagina. In general if the tumor is localized the prognosis is reasonably good. Embryonal rhabdomyosarcomas of the bladder show more superficial involvement and have a better prognosis than prostatic lesions which tend to be more deeply invasive. In one review, 24% of the bladder tumors showed widespread invasion with a survival of 70% after radical surgery, radiation, and chemotherapy. This is compared to prostate rhabdomyosarcomas 40% of which showed extensive local spread. The survival rate after similar combination therapy was only 50%.

Embryonal rhabdomyosarcoma may present as a massive bulging from various orifices but bleeding is not uncommon. The trunk and paratesticular lesions usually present as an enlarging mass. Erosion of bone and metastasis to lung and liver as well as lymph nodes can occur. One clinical staging system is as follows:

- I. Localized, completely resected
 - A. Confined to muscle or organ of origin
 - B. Spread to contiguous part
- II. Localized—residual microscopic disease
 - A. Lymph nodes negative
 - B. Lymph nodes positive (resected)
 - C. Lymph nodes positive (not resected)

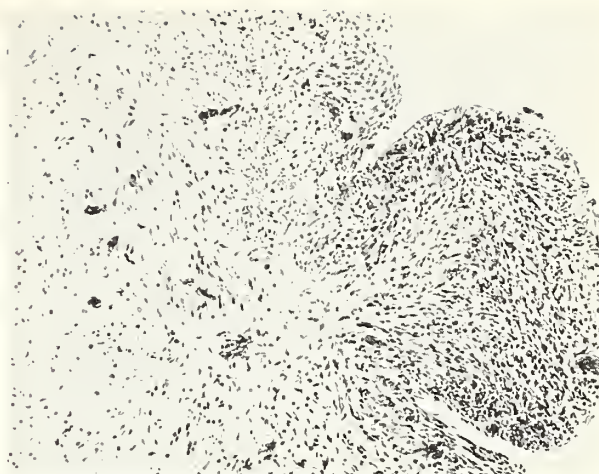


Figure 2
Polypoid lesion with cambium layer 40X.

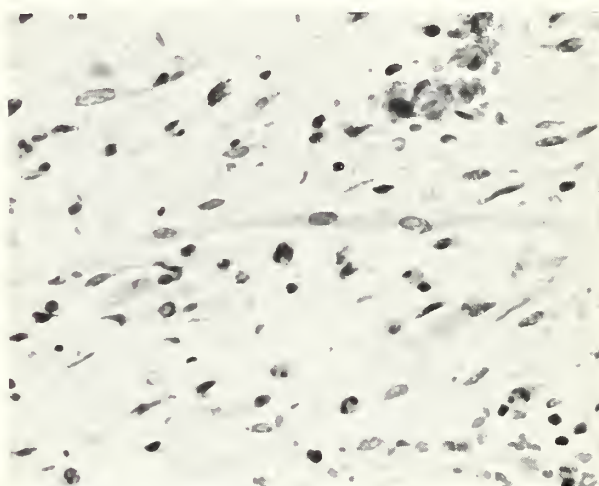


Figure 3
Strap cells typical of rhabdomyosarcoma 430X.

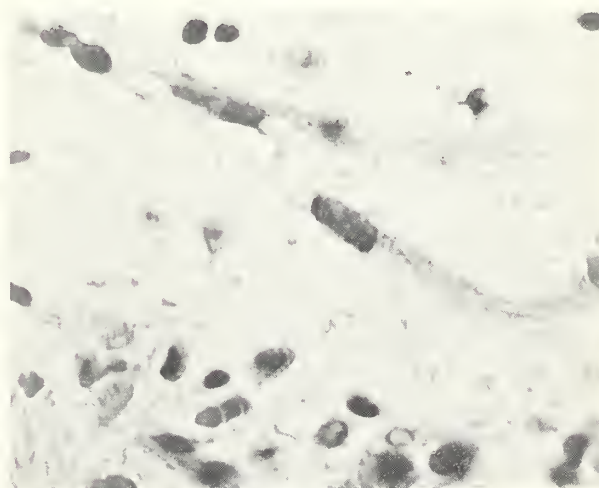


Figure 4
Oil immersion demonstrates cross striations.

- III. Local or regional disease with grossly incomplete resection
- IV. Metastatic disease

DR. THATCHER: I would like to reiterate what Dr. Hoskins has said in regard to the relative infrequency of the tumor. Therefore, it has been difficult to get a clear picture of treatment from the past literature. The tumor also occurs in a large variety of sites and extensive experience with any one site is meager. Several years ago, a large national intergroup rhabdomyosarcoma study was started. This involved approximately 50 or more major pediatric oncology centers. The group was organized to obtain enough cases to evaluate treatment data with large enough numbers to become statistically significant. The average accrual rate of new cases in this group was approximately 11 per month. Children between ages 2 and 7 do better than younger or older children with embryonal rhabdomyosarcomas. This national group found the best prognostic indicator was clinical staging. Their classification is slightly modified from the one given by Dr. Barlow. Stage I deleted spread to contiguous parts. It was felt that if the lesion was localized and completely resected, the resection margins were free and the lymph nodes were not involved, one could classify this lesion as stage I. Stage II was defined as a grossly resected tumor but with microscopically residual disease. In other words, the surgeon thought that he has resected the tumor grossly. Regional nodes could be involved. Stage III represented grossly residual disease with incomplete resection and stage IV was obvious metastatic disease. The five year survival was 90-100% survival in stages I and II, 60% in stage III, and 40-50% in stage IV. Therapy included debulking surgery, chemotherapy and/or radiation. This combination therapy, compared to previous radical surgery alone, appears to be much more effective as noted by Dr. Hoskins.

Because micrometastases rather than locally recurrent disease, seem to be the problem in failure to cure this disease, chemotherapy has played a greater role in treatment of embryonal rhabdomyosarcoma. The combination of drugs generally used includes actinomycin D., vincristine, and cyclophosphamide. The national group is also studying the addition of adriamycin to these combinations and whether it is best to use cyclophosphamide as continuous therapy or intermittent pulse therapy. We gave weekly doses of vincristine for 12 weeks and an initial course of 5 days of actinomycin D and repeated this at three month intervals. Cyclophosphamide was added after approximately three months of therapy and given daily.

One of the unanswered questions in this disease is what is the optimal amount of surgery. I believe, in this patient, that the extent of surgery performed was the best choice. Others have questioned the role of radical surgery and believe it is probably not the treatment of choice. This child has been followed almost two years. We have elected not to give the child radiotherapy. Many experts recommend the use of radiotherapy but it is controversial in very young children since complications from pelvic radiotherapy are numerous. This child has done extremely well without evidence of recurrence for almost two years. We cannot say yet whether this patient will continue to do well because, in some cases, widespread tumor has recurred after 2-3 years.

DR. HOSKINS: Isn't it true that some cases of embryonal rhabdomyosarcoma have apparently been cured by chemotherapy alone?

DR. THATCHER: Possibly but the data is meager in this regard. We may find that chemotherapy alone can be used for the treatment of certain stages of this disease but that is yet to be determined and is not currently recommended.

DR. HOSKINS: I would also like to point out that embryonal rhabdomyosarcomas of the genitourinary tract behave differently than similar tumors located elsewhere in the body.

DR. THATCHER: Yes, this is certainly true. The cure rate of embryonal rhabdomyosarcoma of the orbit is 80% with radiation alone. This tumor also would behave differently than a rhabdomyosarcoma located in an extremity. The location of the tumor must be taken into consideration and each site must be considered separately. Staging must also be employed, of course.

DR. NORDSTROM: I have been associated with an institution that uses a slight modification of this staging system. Clinical stage I was a localized neoplasm which was either subcategorized into subdivisions a and b—completely resected and incompletely resected because of anatomic consideration. An example would be a lesion of the orbit where resection would necessarily be limited. The same would be true of lesions of the nasopharynx or ear. Stage II was localized disease with microscopic residual tumor, stage III was grossly incomplete resection, and stage IV represented metastatic disease. Our radiotherapy treatment was modified according to this schema. In patients with completely resected localized disease, no radiotherapy was

given. Lesions with microscopic or grossly residual disease (stage Ib, II, and III) were given radiotherapy. Chemotherapy was applied in all cases. Our radiation dose was 6000 rads given over a 6-week period. This dose of radiation in a child can produce a number of complications. One of the complications is cystitis which is not only a complication of the radiation but also of the cyclophosphamide. Also bone growth in the pelvis can be altered by radiation. The combination of radiotherapy and actinomycin D can also produce skin reaction. The place of radiation in stage IV (metastatic disease) is strictly palliative. The overall survival rate corresponded to the chemotherapy group. Stage I yielded an 85% survival; stage II and stage III, 69%.

DR. THATCHER: Recent statistics which I have received lately from the national study group have suggested that radiation therapy can be reduced from 5000 to 3500 rads with no sacrifice in survival. Although we don't know for certain, it may be that combination chemotherapy will enable us to further reduce the dose of radiation; and, in cases where there is only microscopic residual disease, possibly dispense with it.

DR. BARLOW: What would you do if this patient had a local recurrence?

DR. THATCHER: I would think that radiotherapy would be indicated and we might ask Dr. Hoskins to consider a further resection. We would also use different chemotherapy such as adriamycin since we would not expect the patient to respond to the standard chemotherapy if he had recurrent disease after this length of time.

***DR. DORENCE ENSBERG:** How long do you maintain chemotherapy?

DR. THATCHER: We presently continue chemotherapy for two years and then stop if there is no evidence of any recurrence. The usefulness of longer duration chemotherapy has not been demonstrated.

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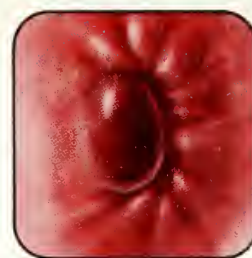
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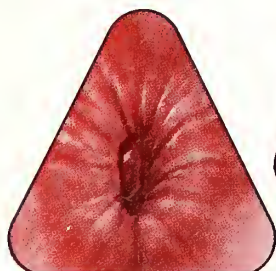
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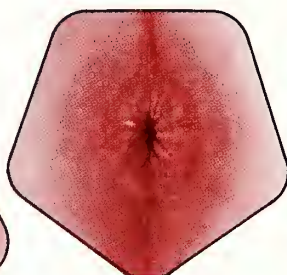
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Urology Postgraduate Conference, U. of Iowa Hospitals, Iowa City, IA, Sept. 28-29. Fee: \$75. 8.5 hrs. AMA Category 1 credits. Contact: Off. of Cont. Med. Ed., U. of Iowa College of Med., 285 Med. Labs, Iowa City, IA 52242.

October

Annual Internal Medicine Review, Mayo Mem. Aud., U. of MN, Minneapolis, MN, Oct. 3-5. Fee: \$155. 21 hrs. AMA Category 1 credits. Contact: Cont. Med. Ed., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Allergy for the Primary Care Physician, Creighton Univ. School of Medicine, Omaha, NE, Oct. 5-6. Fee: \$70.00. 13 hrs. AAFP & AMA Category 1 credits. Contact: Div. of Cont. Med. Ed., Creighton Univ. School of Med., Omaha, NE 68178.

Cardiology Today, U. of Iowa Hospitals Iowa City, IA, Oct. 8-11. Fee: \$275. 30 hrs. AAFP & AMA Category 1 credits. Contact: Carl White, MD, Cardiovascular Div., U. of Iowa Hospitals and Clinics, Iowa City, IA. Phone: (319) 356-3413.

AMA Regional CME Meeting, Llikai Hotel, Honolulu, HI, Oct. 8-12. 25 hrs. AMA Category 1 credits. Contact: AMA, 535 N. Dearborn St., Chicago, IL 60610.

Hypertension in Family Practice, Georgia World Congress Center, Atlanta, GA, Oct. 11. 3 hrs. AAFP & AMA Category 1 credits. Contact: World Health Info. Services, Inc., 505 Park Ave., New York, NY 10022. Phone: (212) 751-5737.

Sexual Attitude Reassessment Seminar, Research East Bldg., U. of Minn., Minneapolis, MN, Oct. 12-13. Fee: \$150.00. 16 hrs. AMA

Category 1 credits. Contact: Cont. Med. Ed., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Basic Principles of Management of the Poisoned Patient, St. Paul Ramsey Med. Ctr., St. Paul, MN, Oct. 17. Fee: \$25. 5 hrs. AMA Category 1 credits. Contact: Cont. Med. Ed., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Fourth Annual Perinatal Conference, Sheraton Inn, Aberdeen, SD, Oct. 18-19, 13 AMA Category I credit hrs., 11 AAFP hrs. Contact: Margo Varcoe, R.N., Exec. Sec., SD Perinatal Assoc., 1100 S. Euclid Ave., Sioux Falls, SD 57105. Phone: (605) 339-6578.

Central Nervous System Disease Symposium, Hennepin County Med. Ctr., Minneapolis, MN, Oct. 19. Fee: \$50. 6 hrs. AMA Category 1 credits. Contact: Cont. Med. Ed., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

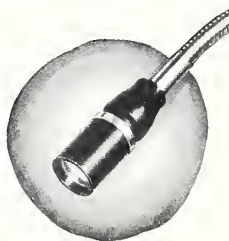
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**Reimplantation of Nearly Amputated
Upper Extremity: Long-term Followup**

**Clinicopathological Conference
Seven Month Old Child with Fever,
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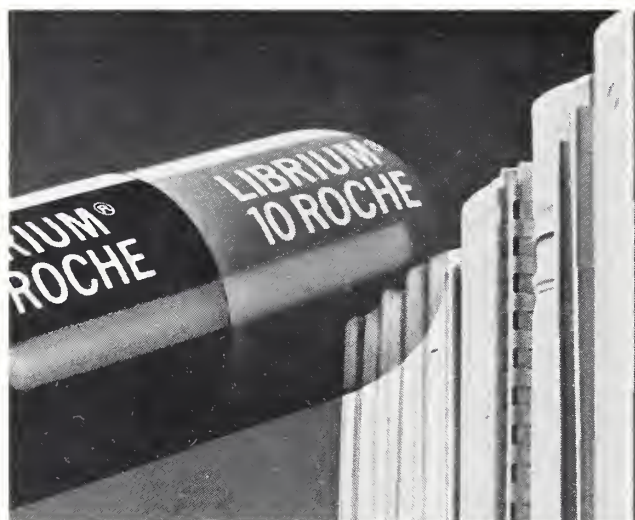
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SCIENTIFIC ARTICLES

- 11 Reimplantation of Nearly Amputated Upper Extremity:
Long-term Followup
Robert E. Van Demark, M.D., FACS
- 19 Clinicopathological Conference Seven Month Old Child with
Fever, Lethargy and Bulging Fontanel
Thomas Huber, M.D.
John F. Barlow, M.D.

FEATURES

- 29 President's Page
- 30 X-Ray Case of the Month
- 33 South Dakota AFP Chapter News
- 35 This Is Your Medical Association
- 40 Laboratory Aids
South Dakota Pathology Page
- 42 Future Meetings

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Reimplantation Of Nearly Amputated Upper Extremity: Long-Term Followup*

Robert E. Van Demark, M.D., F. A. C. S.**

ABSTRACT

A white male, age 56, suffered an almost complete amputation of the right upper extremity, resulting in a white avascular and anesthetic forearm and hand. Prompt cleansing, debridement, bone, soft tissue and vascular repair within four hours of injury restored good pulsations in the distal extremity. Secondary skin closure and late neuroorrhaphy were subsequently carried out. The late result showed rather marked restriction of elbow motion, excellent vascularity and a good functional right hand.

Replantation of the upper extremity was popularized in the previous decade by the national press at the time of the brilliant work of Malt and his co-workers⁽¹⁾ at the Massachusetts General Hospital where the upper limb of a ten-year-old boy was reattached with a viable result. A ten year followup⁽²⁾ (the longest reported at that time) revealed a functioning hand following multiple surgical procedures and transplants.

In 1974 O'Brien and his associates⁽⁴⁾ in an article entitled "Major Replantation of the Upper Limb" reported 7 cases of complete or incomplete amputations of the upper extremity with an overall survival rate of more than two-thirds; two incomplete amputations failed to survive.

O'Brien et al⁽⁴⁾ stated that replantation was not advised in patients over fifty years. In 1977 Morrison et al⁽³⁾ stated that because of the large muscle content in amputations above the mid forearm level, replantation yields poor function or there is failure to survive.

Today it would seem desirable to record the long term result of such a case in a fifty-six year male who returned to his regular work duties, has since retired and who kindly agreed to be present for this presentation today.

*Patient and paper presented May 6, 1978 at the 25th Annual Meeting of South Dakota Chapter of the American College of Surgeons at Sioux Falls, SD.

**Orthopedic Surgeon, Orthopedic and Fracture Center, P.A., Sioux Falls, SD.

CASE REPORT

A white male, age 56, was seen on October 13, 1967 in consultation at the emergency room at McKennan Hospital because of a nearly amputated right arm. The patient, the foreman of the beef kill in a local packing plant, caught his right arm in a lift chain while trying to repair it. He had bled profusely during the period when his broken arm was engaged in the chain and suspended only by soft tissue attachments on each side which were maintained in its careful removal.

On examination (Fig. 1) the extremity was flail distal to the elbow, white and pulseless with only soft tissue attachments medially and laterally between the deep volar and dorsal lacerations which extended to the bone anteriorly and into and through the bone posteriorly (Fig. 2). Bleeding from the divided brachial artery had been promptly controlled at the emergency room by Doctor Calindo, the intern who had ligated the tip of the proximal brachial artery. The patient's general condition was good other than for an acute blood loss which required seven units of whole blood to stabilize.

Roentgenographic examination showed extensive bone and soft tissue injury (Fig. 2).

Following intravenous fluids and stabilization with blood, the extremity was thoroughly cleansed with soap and water and debrided. The ulnar nerve was bruised but continuous as was the distal radial nerve. The anterior Monteggia fracture dislocation was reduced and stabilized with an intramedullary Rush pin in the ulna (Fig. 3). Attention was then



Figure 1

Division of all the anterior soft tissue structures down to the humerus with retraction of the brachial artery ends. Hand and forearm white and pulseless.



Figure 2

Posterior laceration through the ulnar shaft with anterior Monteggia fracture-dislocation.



Figure 3

Result following reduction and stabilization with intramedullary Rush pin in ulna.

turned to the brachial artery, the ends of which could be approximated by flexing the elbow. After repair of the soft tissues, the arterial ends were sutured with 6-0 silk, and a full strong pulse appeared at the wrist with restoration of good capillary circulation. Circulation of the forearm and hand was re-established just within four hours of injury.

The skin margins were left open for delayed closure and a posterior plaster splint was applied for immobilization over voluminous dressings. Profuse antibiotic coverage was used, but no anticoagulant therapy. Closure of the skin was later performed after no evidence of infection occurred. Subsequent excision of the neuroma and suture of the median



Figure 4

Dorsal view of elbow showing final post-operative extension and circumferential scarring.



Figure 5

Extension of the elbow and digits achieved.



Figure 6

Flexion of the elbow and digits.



Figure 7

Opponens action of the thumb after median nerve suture. Patient has 2 point discrimination of 10 mm. Radial and ulnar pulsations, capillary circulation and venous return were good.

nerve was done with a good functional result. He now has 2 point discrimination in the tip of the index finger of 1 cm., and good opponens function (Fig. 7).

The patient returned to his previous occupation and duties. His hand does not feel "normal" but he can do everything with it. His elbow shows considerable limitation of motion as follows: flexion 62°, extension 130°, pronation-supination 20°. (Figs. 4, 5, 6)

DISCUSSION

Pain and swelling have not been a problem in these cases as emphasized by Malt.⁽²⁾ Sensitivity to cold does occur regularly.

The more distal the injury, the better is the prognosis. The ideal patient is a younger individual with a clean amputation at or below the mid-forearm level. Malt et al⁽²⁾ have suggested that replantation may be justified even if the circumstances are not

ideal, provided that an unsuccessful outcome is promptly reconverted into an amputation.

The long term results in this case lend encouragement to the salvage attempt in the older individual with an injury to the dominant extremity.

Re-establishment of the circulation in this patient's forearm and hand within a period of four hours following the injury favorably affected the prognosis.

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The Maker

Examining a Few Myths About Prescribing.

Increasing pressure is being put on the practicing physician to prescribe drugs generically. You are told that brand-name products are universally "expensive" and generic versions are relatively "cheap." To make this case, the most extreme (rather than typical) price differentials are cited. Thus, consumers are led to believe that such differentials are commonplace. Even your knowledge and your motives as a physician are questioned.

Understandably, these views have created myths. We think it's time to examine them in the light of all the facts and ramifications.



MYTH: There are no differences in quality and performance between brand-name products and their generic counterparts. The corollary is that there are no differences among products made by high-technology, quality-conscious, research-based companies and those made by commodity-type suppliers.

FACT: The Food and Drug Administration does a good job in monitoring a generally excellent drug supply. Still, it has nowhere near the resources to guarantee the quality and bioavailability of all marketed products at any given time. Just a few months ago, for example, it noted that batches of tetracycline HCl capsules which met official monograph requirements were

not bioequivalent to a reference product. As you know, there is substantial literature on this subject affecting many drugs, including such antibiotics as tetracycline and erythromycin. The record on drug recalls and court actions affirms strongly that there are differences among pharmaceutical companies and their products. Research-intensive companies have far better records than those that do no research and may practice minimum quality assurance.

MYTH: Industry favors only "expensive" brand names and denigrates all generics.

FACT: PMA companies make 90 to 95 percent of the drug supply, including, therefore, most of the generics. Drug nomenclature is not the important point; it's the competence of the manufacturer and the integrity of the product that count.

Seven Month Old Child With Fever, Lethargy And Bulging Fontanel

Thomas Huber, M.D.*
Discusser

John F. Barlow, M.D.**
Pathologist-Editor

Case No. 789013

This seven-month-old male infant was admitted to Sioux Valley Hospital for lethargy, fever, and bulging fontanel.

This child was well until the day prior to admission when he developed lethargy and limpness followed by vomiting. He was taken to a local physician who gave oral erythromycin but his temperature rose to 105° shortly thereafter. He was admitted to another hospital where he was given sodium luminal, oral erythromycin, aspirin, and lactated ringer's solution subcutaneously. There were no seizures. The baby was transferred. Past history revealed a possible ampicillin allergy. The baby went limp in her arms after an ampicillin shot a few months previous.

PHYSICAL EXAMINATION: Pulse 176/minute and regular; respirations 60/minute, temperature 102.4°F; the baby was a listless, lethargic, but a somewhat irritable child. The anterior fontanel was visibly bulging and tense. Examination of the eyes, ears, and mouth showed no abnormality. The neck was supple but the baby cried with flexion of the neck. The lungs were clear to auscultation and percussion. Examination of the heart revealed a regular rhythm with no murmurs. Examination of the abdomen showed no tenderness, organs, or masses. Examination of the genitalia and extremities were within normal limits.

LABORATORY DATA: Hemoglobin on admission 11.1 gm/dl, red count 4.07 million/mm³, hematocrit 31 vol/dl, mean corpuscular hemoglobin 27 micromicrograms, mean corpuscular volume 77 cubic micra, mean corpuscular hemoglobin concentration 35%, white count 6500/mm³ with

14% segmented neutrophils, 27% neutrophilic bands, 1% eosinophils, 57% normal lymphocytes and 1% monocytes. The platelets were normal in number and morphology and the red cells were normochromic, normocytic. Urinalysis yellow, clear, specific gravity 1.020, pH 7.0, negative for protein, glucose, bile and hemoglobin, trace ketone bodies; sediment 3 to 4 white cells/hpf, 2 to 3 red cells/hpf; blood pH 7.33, PCO₂ 42 torr, CO₂ content 23 mm/L, sodium 137 meq/L, potassium 4.8 meq/L, chloride 103 meq/L. Blood sugar 81 mg/dl, cerebrospinal fluid colorless; 78 rbc's, 86 wbc's with 86% segmented neutrophils and 14% mononuclear cells, protein 146 mg/dl, sugar 1 mg/dl. A counter immunoelectrophoresis revealed a positive test for hemophilus influenzae, type B and negative tests for streptococcus, group B; meningococcus, and pneumococcus. Gram stains of the spinal fluid showed no bacteria. Cultures of the spinal fluid revealed hemophilus influenza, type B as did a blood culture. The organism was resistant to ampicillin and sensitive to carbencillin, chloramphenicol, tetracycline, gentamicin, kanamycin and tetracycline. Beta lactamase was produced by the organism. Serum iron 104 mcg/dl, iron binding capacity 289 mg/dl and percent saturation 36%.

The spinal fluid 8 days after admission revealed 3 red cells, 102 white cells with 24% segmented neutrophils, and 76% mononuclear forms, protein 122 mg/dl, sugar 35 mg/dl. A computer tomogram of the brain was negative but a brain scan showed widening of the area about the sagittal sinus toward the right.

DR. HUBER: The diagnosis in this patient would be acute bacterial meningitis caused by hemophilus influenzae (H. flu), type B, the most common cause of bacterial meningitis in this age group. The discussion that follows will be a general discussion of acute bacterial meningitis and will emphasize pertinent aspects of this case.

*Resident in Family and Community Medicine, Sioux Falls, SD.

**Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Professor of Pathology, School of Medicine, University of South Dakota.

Acute bacterial meningitis varies as to etiology and clinical presentation depending on the age of the patient. Neonatal meningitis (birth to 1 month) is usually caused by Group B streptococcus or *E. coli*. Infant meningitis (1 month to 1 year) is most often caused by *H. flu*, type B. Older children and adults generally have *Neisseria meningitidis* (meningococcus) or streptococcus pneumoniae (pneumococcus) as the causative organisms of meningitis. The geriatric population is most susceptible to the gram negative rods, other streptococci and the pneumococcus.

Neonatal meningitis has an incidence of 40-45/100,000 live births and approximately 2/3 of the cases occur in hospitals. Maternal factors associated with this problem are traumatic delivery, prolonged labor, prolonged rupture of membranes, perinatal infection or antibiotic therapy which has selected resistant bacteria. Fetal factors include prematurity, low birth weight (less than 2500 grams), generalized sepsis or lack of immunologic experience and/or competence. Other factors include contaminated equipment and carrier personnel. *E. coli* is the causative organism in about 40% of cases, Group B streptococcus in 15-20%.

H. flu meningitis has its main incidence between 3 months and 3 years of age and has maximum incidence between 6 to 8 months of age. The frequency then decreases slowly after 12 months until the incidence of *H. flu*, meningococcus and pneumococcus is approximately equal at 2 years of age. *H. flu* is responsible for 60% of postneonatal bacterial meningitis. It is felt there is a temporal correspondence of the onset of vulnerability to the disease caused by *H. flu* with the waning of maternally acquired level of humoral antibody bactericidal for the organism. The present incidence is 50/100,000. If one finds *H. flu* as an etiologic agent in a patient over 10 years of age, one should suspect a parameningeal focus or a possible immunoglobulin deficiency. *H. flu* seems to occur more frequently in the autumn or early winter.

Streptococcus pneumoniae (pneumococcus) assumes importance generally in the toddler age group or older. 2/3 of the cases of bacterial meningitis caused by pneumococcus have underlying pneumonia or a parameningeal focus of infection. This is the most common organism seen in patients with recurrent bacterial meningitis. Most of these patients have a history of recent or remote head trauma with a known or suspected skull and dural defect often in the ear or cribiform plate.

Meningococcus is the only organism to cause epi-

demio bacterial meningitis. There is an observed periodicity to the epidemics—about every 10 years. The organism is seen in military camps and barracks where overcrowding and fatigue are present. The meningococcus is spread from person to person by nasopharyngeal secretions. The asymptomatic nasopharyngeal carrier rate varies from 1% in nonepidemic situations to over 70% in epidemics.

The pathogenesis of acute bacterial meningitis can be complex; however, 4 major pathways are generally discussed.

1. Bacteremia with focal implantation is the most common pathway. The disease is essentially a metastatic blood borne infection. This holds true for pneumococcus with an underlying lobar pneumonia or endocarditis, *H. flu* with otitis media or other respiratory infection and meningococcus with nasopharyngeal carrier state or mild rhinopharyngitis. It should be noted that staphylococcus aureus and the enterobacteriaceae are the most common causes of bacteremia in humans but are not common causes of meningitis. The predilection of the meningococcus, pneumococcus and *H. flu* for the meninges is unknown. Interestingly all possess a capsule as do *Escherichia coli* (K1 capsule) and Group B streptococci which cause newborn meningitis.

2. Lymphatic drainage from infected sites such as middle ear space, mastoids or sinuses.

3. Direct implantation of organisms occurring with trauma such as skull fractures involving the sinuses e.g. surgery or bullet wounds to the head.

4. Contiguous spread of organisms from upper respiratory infections or facial infections.

The pathology of the meningitis is usually very distinct; unless, of course, the time from onset of disease to demise of the patient is very short. The brain surface is covered with a layer of purulent matter consisting of polymorphonuclear leukocytes, fibrin, bacterial clumps and red cells. There is a diffuse vasculitis with areas of necrosis of the vascular walls and thrombosis of arteries and venous sinuses. This leads to decreased perfusion and cerebral edema. Fibrinous exudates develop which can block the flow of cerebrospinal fluid from the meningeal foramina resulting in obstructive hydrocephalus. Cranial nerves, especially II, III, IV, VI, VII, and VIII, can be involved by the inflammatory process with resultant paresis.

The clinical manifestations of acute bacterial meningitis are independent of the etiologic agent. Symptoms and signs result from infection and inflammation first, and later from increased intracranial pressure.

The neonate with meningitis is usually well at birth but then shows a general diminution of function with poor tone, weak cry and sucking, followed by decreased movement. Vomiting or diarrhea may be present. Fever, or conversely hypothermia, may be seen as well as jaundice. The infant may present acutely with apnea, seizures, or even cardiovascular collapse. A full fontanelle is a variable finding and the neck is usually supple.

In the infant, the bulging fontanelle is the most significant physical finding. Nuchal rigidity may or may not be present. There may be fever, vomiting, hyperirritability, convulsions, or a "high pitched cry".

In children and adults, the findings are more classic. Usually present is a headache described as severe, bursting, unremitting, diffuse and mainly frontal often radiating down the neck and into the back. Fever with chills and vomiting is seen. A convulsion may be the initial manifestation. A history of preceding respiratory or gastrointestinal symptoms is variable. The level of consciousness of the patient with bacterial meningitis varies with the severity. Initially, there is irritability which progresses to delirium and mania, then to drowsiness, stupor and finally, coma (if allowed to proceed without intervention). Signs of meningeal irritation are present and manifest as 1) cervical rigidity which is elicited as Brudzinski's sign in which rapid neck flexion brings about knee flexion, 2) thoracolumbar rigidity in which the patient maintains a posture of fixed hyperextension of the spine—referred to as opisthotonus or "tripodding", 3) Kernig's sign in which with the thigh and knee flexed, knee extension is met by much resistance due to hamstring spasm, 4) exaggerated reflexes which are symmetrical and hyperactive. Petechiae or purpura may be a sign of meningococcemia. A congenital dermal sinus should raise a suspicion of *E. coli* infection which has gained access to the subarachnoid space through a communication between the sinus and the subarachnoid space.

Again, classic signs and symptoms help but these may not be present in every patient. Therefore, any patient suspected of having meningitis needs to have a lumbar puncture with cerebrospinal fluid analysis. Normal values for lumbar cerebrospinal fluid are given below:

Glucose	newborn	20-40 mg/dl
	infant/child	70-90 mg/dl or 1/2-2/3 of the serum glucose
	adult	50-80 mg/dl
Protein	newborn—1 month	20-120 mg/dl
	thereafter	15-45 mg/dl

In bacterial meningitis, the cell count is usually greater than 1000/mm³ in untreated cases. The count may be normal very early. Therefore, a repeat spinal tap may be needed to show a cellular response. A gram stain may demonstrate the organism. Pneumococci are usually easily seen but meningococci and *H. flu* organisms are seen with difficulty. A culture is absolutely necessary. The cerebrospinal fluid glucose is usually decreased. This is felt to be due to an interference with the transport of glucose from the blood to the cerebrospinal fluid which is mediated by both a passive transport system and a carrier-mediated system. Originally it was thought that the glucose level was decreased because it was consumed by the bacteria during growth. However, this probably plays only a minor role in the glucose reduction. The cerebrospinal fluid protein is elevated due to three processes: 1) leakage of serum proteins into the cerebrospinal fluid because of altered membrane permeability; 2) release of proteins from the meninges as products of inflammation (transudation or exudation) and 3) leukocyte breakdown.

The cerebrospinal fluid lactic acid level can be helpful in differentiating bacterial from viral meningitis. In most studies, there is no overlap between lactate levels in patients with bacterial or tuberculous meningitis versus those patients with nonbacterial (viral) meningitis. Different sources quote different values—Controni et al use lactate levels greater than 25 mg/dl as indicative of bacterial etiology; Brook et al use 35 mg/dl as the cut off. Other causes of elevated cerebrospinal fluid lactic acid levels include a moderate increase in intracranial pressure and/or significant decrease in cerebral blood flow causing a shift from aerobic to anaerobic metabolism from whatever etiology; hypercapnia; hydrocephalus; brain abscess or cerebral ischemia.

A more recent laboratory aid to be developed is the use of countercurrent immunoelectrophoresis (CIE). This utilizes the concept of placing a solution with a high titer of antibody to a specific antigen (organism) in contact with that antigen and observing a precipitin reaction using CIE as described by Hoeprich. This procedure is not readily available

Cell count	premature/newborn	018 wbc/hpf
	infant/thereafter	0-8 wbc/hpf

at most smaller hospitals but it offers an advantage of rapidity of diagnosis as results are generally available within one hour. At present, it is only useful in detecting *H. flu*, meningococcus, and pneumococcus. Shown below is a table comparing results of cerebrospinal fluid analysis in different conditions which should be included in the differential diagnosis of acute bacterial meningitis.

Meningismus is clinically evident meningeal irritation but without cerebrospinal fluid abnormalities except possibly a slightly elevated protein. This can be seen in acute, specific childhood febrile illnesses, influenza, pneumonia, typhoid fever and acute rheumatic fever.

Other conditions to be included in the differential diagnosis include shigellosis, pyelonephritis, a renal abscess, the acute abdomen, local neck or pharyngeal infections—such as cerevical adenitis, retropharyngeal or peritonsillar abscess, and subdural empyema. In the infant, tetracycline, hypervitaminosis A and steroids may produce a bulging fontanelle.

The complications which can arise from acute bacterial meningitis are many and varied. A clinical picture of peripheral circulatory collapse with extensive disseminated intravascular coagulation, adrenal hemorrhage and failure is termed the Waterhouse-Fridericksen syndrome and is usually associated with meningococcemia. However, this can be seen with any of the common organisms causing meningitis. Polyarthrits and migratory arthritis is usually a result of meningococcus. Cranial nerve dysfunctions, especially cranial nerves II, III, VI, VII, and VIII may be transient or permanent. Cerebral arteritis or phlebitis can give rise to focal seizures or persistent hemiparesis. Obstructive hydrocephalus may develop as previously mentioned.

Subdural effusion develops in 6-17% of the pediatric patients, usually in cases of *H. flu* or pneumococcal meningitis. The pathogenesis of this is uncertain and the fluid is usually sterile. It should be suspected when there is prolonged fever, recurrent

or persistently bulging fontanelle, convulsions or focal neurological signs such as focal paralysis. The diagnosis and treatment involve the same procedure, namely subdural needle tap with aspiration. Continued accumulation after 2 weeks of conservative management generally means surgical excision of the sac is necessary.

The treatment for acute bacterial meningitis involves the usually supportive measures as needed. Antibiotics are selected based upon the causative organism. In the neonate, no strict guidelines have been established. Therefore, I will not attempt to be complete. Ampicillin and an aminoglycoside are often utilized in neonatal meningitis. In infants with *H. flu*, initial therapy should be both ampicillin as well as chloramphenicol intravenously since strains of ampicillin resistant *H. flu* are more common today. Ampicillin is started as a 50 mg/kg bolus intravenously, then 200 to 300 mg/kg/24 hours given every 6 hours. Chloramphenicol is started as a 50 mg/kg intravenous bolus, then 100 mg/kg/24 hours are given every 6 hours. Continuance of either drug hinges on the antibiotic susceptibility of the organism. Ampicillin should be given for a minimum of seven to ten days after the patient is afebrile provided the repeat cerebrospinal fluid analysis is normal. Maximum recommended chloramphenicol dosage is 4 grams/day. Pneumococcal and meningococcal meningitis are both initially treated with intravenous penicillin G. Chloramphenicol is indicated for penicillin resistant organisms. Penicillin G dosage in children under 2 is 100,000 to 200,000 units/kg/day and in older children and adults 100,000 to 400,000 units/kg/day given every 4 hours. In adults this is usually 10 to 20 million units per day intravenously. Treatment should continue for 10 to 14 days with repeat cerebrospinal fluid analysis to indicate the time of cessation of therapy. Chloramphenicol dosage is the same as that for *H. flu*. Adults should receive 3 gms per day.

Prophylactic antibiotics have been used in meningococcal disease to try to eliminate nasopharyngeal

Table I

Disease	Cells	Glucose	Protein
Acute bacterial meningitis	↑ (polys)	↓	↑
Tuberculous meningitis	↑ (lymphs)	↓	↑
Aseptic meningitis (viral)	↑ (lymphs)	normal	↑/normal
Brain abscess/tumor	normal/ ↑ (lymphs)	normal	↑
Lead encephalopathy	normal/ ↑ (lymphs)	normal	↑
Meningismus	normal	normal	normal

carriage and to prevent epidemics. Some success has been reported using rifampin with dosage varying with age: 5 mg/kg every 12 hours orally for children less than 1 year old, 10 mg/kg every 12 hours for age 1 to 12 years, and 600 mg every 12 hours in adults. Minocycline has also been investigated for prophylaxis in meningococcal meningitis. However, vestibular side effects have prevented its widespread use.

Prevention of bacterial meningitis through immunization is coming more to the forefront today and development of vaccines is proceeding around the world. All of the 5 major organisms involved bear polysaccharide capsules on their surfaces. These are the antigenic components used in typing these organisms. The 5 organisms are Group B streptococcus, type III; *E. coli* (K1 capsule); hemophilus influenza, type b; pneumococcus (several serotypes); and meningococcus (several serotypes). For each of these, the polysaccharide capsule is a virulence factor and antibodies directed to the capsule confer immunologic protection. Pneumococcus has 83 different types but 90% of infections, including lobar pneumonia or meningitis, are due to only 18 serotypes. The Pneumovax® vaccine marketed commercially contains 14 serotypes and covers 80% of the serotypes causing pneumococcal disease. Meningococcus types A, B and C cause most of the meningococcal disease. A and C polysaccharides are excellent antigens in people over 2 years of age. They have a low toxicity and have been tested in over 120,000,000 people worldwide. Type B polysaccharide has proved disappointing as an immunogen at any age. A study in Finland has found the type b polysaccharide of hemophilus an effective antigen in preventing meningitis in children over 18 months old but it generates insufficient immunogenicity in children under 18 months of age. Sera of mothers who have delivered infants who subsequently developed type III Group B streptococcal sepsis have low levels of antibodies to this antigen when compared to controls. A vaccine for this organism is still very investigational at present. The K1 antigen of *E. coli* is chemically identical to the Group B meningococcal antigen and therefore has had the same unsuccessful immunologic trials due to poor antigenicity.

The philosophy behind immunizations for prevention of bacterial meningitis is three-fold: 1) prevent epidemic meningitis—this is usually due to meningococcus. Therefore, the vaccine containing meningococcus A and C antigen should be administered to the population at risk in the area of the epidemic; 2) prevent endemic meningitis—this

should utilize antigens to prevent *H. flu* type b, meningococcus, and pneumococcus and should be given to children between 3 and 6 months of age and repeated at as of yet undetermined intervals until school age when immune response becomes long-lived; 3) prevent neonatal meningitis—to ensure that pregnant women have very high levels of antibodies to Group B streptococcus (especially type III) and antibodies protective against *E. coli* K1. Logically, one would immunize in the third trimester. However, debate is heated on this point. Those in favor of such a course of prenatal immunization say that the vaccine then becomes part of routine prenatal care, the antibodies are at peak levels at the time of delivery, and you would avoid vaccinating early in pregnancy. Those opposed bring up the questions of possible teratogenicity of the vaccines, induction of immunologic paralysis to the fetus and a possible abortifacient effect.

The preventive medicine aspects of meningitis are evolving slowly and time will tell if this will have an effect on disease.

The prognosis in anyone with bacterial meningitis depends again on several factors: 1) age; 2) organism; 3) severity of disease; 4) duration of disease before treatment and 5) antibiotic susceptibility of the organism. The prognosis is worse in the very young and very old, is worse with the coliforms and group B streptococcus, and is worse in the presence of the Waterhouse-Fridericksen syndrome. 50% of neonates who recover have a significant motor or intellectual impairment. The complications previously alluded to, if present, affect the prognosis. Even today, the mortality from meningococcus is 5-10%, from hemophilus influenzae about 10%, and from pneumococcus about 15%.

It is all too obvious that early diagnosis and early aggressive therapy is indicated in any case of bacterial meningitis.

In this patient, the management should consist of the use of chloramphenicol with the discontinuance of ampicillin. The toxicity of chloramphenicol must be kept in mind but it is the drug of choice for the present situation.

The additional information given after 8 days of therapy; i.e. the negative computer axial tomogram scan of the brain but a brain scan showing widening about the sagittal sinus toward the right along with the persistently abnormal cerebrospinal fluid analysis, would lead one to believe the child has developed a subdural effusion which should be treated with needle aspiration. Another consideration might be abscess formation. However, subdural effusion is most likely.

Dr. Huber's Diagnosis

Hemophilus Influenzae Meningitis With Subdural Effusion

***DR. W. ALLEN BOADE:** The scan on this patient shows a widened area of increased uptake of radionuclide just below the region of the skull. This represents increased vascularity to the meninges. On the anterior view, there is a asymmetrical area of increased uptake which could represent subdural effusion or subdural empyema. (Fig. 1) Both of these conditions are easily detected because of the increased vascularity associated with the conditions. Other complications of bacterial meningitis include brain abscess, ventriculitis and osteomyelitis of the skull. The first two can easily be detected by the radionuclide brain scan and the later can be detected by the use of a bone scanning agent. This scan is another case demonstrating ventriculitis (Fig. 2).

****DR. JEROME BLAKE:** When a patient is being treated for bacterial meningitis, we follow the patient clinically. If there is persistent fever or such signs as lethargy or alteration of consciousness or if repeat spinal tap continues to show abnormalities, then a brain scan is indicated to look for subdural effusion, subdural empyema, or brain abscess. We have found the brain scan more useful in this regard than the computer axial tomogram.

DR. HUBER: I should have mentioned that 50% of patients who have meningitis develop neurological sequelae. The mortality and incidence of neurological sequelae are much higher in neonatal meningitis than in childhood meningitis.

DR. BLAKE: I think we must treat neonatal and childhood meningitis as two separate diseases. The organisms are different and the prognosis and mortality as well as the treatment are completely different.

DR. BARLOW: I would like to discuss two tests used in this case, which can be helpful in the management of H. flu meningitis. One of these is the counter immunoelectrophoresis or CIE as mentioned by Dr. Huber. This test is performed by placing an antibody (in this case the antibody to H. flu type B) in one well and spinal fluid in the other well of an agarose plate. When electrical current is applied the antigen (hemophilus influenzae organisms in this case) would be driven toward the antibody and



Figure 1
Anterior view shows an area of increased uptake of radionuclide toward right of midline indicating subdural effusion.



Figure 2
Note outline of ventricular system of brain due to ventriculitis.

a precipitin line is formed. The test is useful because frequently the gram stain of the spinal fluid can be negative. When the counter immunoelectrophoresis gives a positive result for an organism, the appropriate treatment can be started before waiting a day or more for the culture results to return. Another helpful property of this test is that one should remember that the gram stain can be misinterpreted. A positive test in the counter immunoelectrophoresis (CIE) can help confirm or correct the interpretation of the gram smear of spinal fluid. We perform the test in this laboratory on spinal fluid or other body fluids for H. flu, type b; streptococcus, Group B; meningococcus, group A and C; and pneumococcus. The antiserum for pneumococcus is a polyvalent

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antiserum. The test can be applied to blood or urine in septicemic infections for these organisms.

Another useful test is the determination of beta lactamase (Penicillinase) activity. Ampicillin resistant strains of *H. flu* type B have been isolated in the last several years. The mechanism of the resistance has been the production of beta lactamase by some strains of this organism. As the test can be performed one day before the usual susceptibility determinations, the physician has information sooner as to whether he is dealing with a strain of organism resistant to ampicillin.

FINAL DIAGNOSIS

MENINGITIS DUE TO HEMOPHILUS INFLUENZAE, TYPE b WITH SUBDURAL EFFUSION

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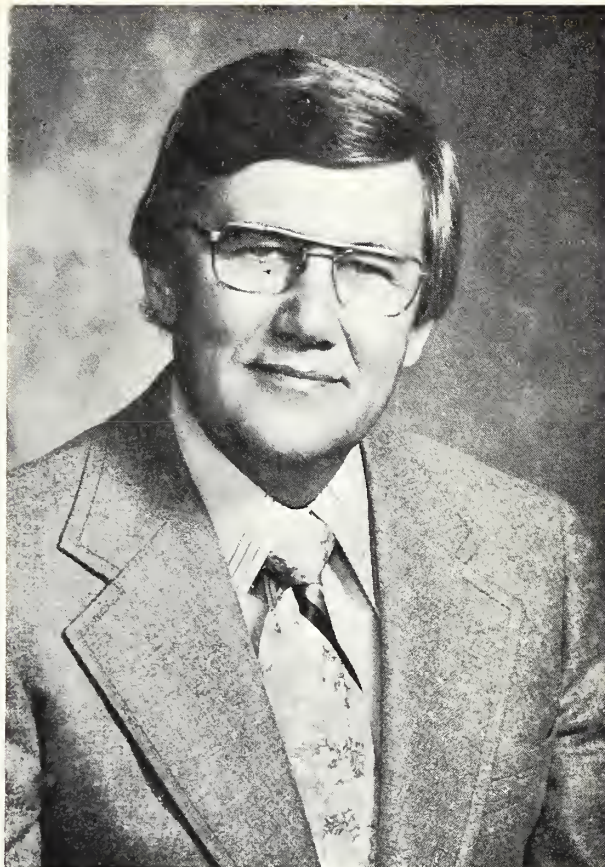
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S D

President's Page



At the Annual AMA meeting in Chicago, your South Dakota delegation pondered many problems that surfaced during the convention, but probably none caused more concern and debate than the AMA's "official" stand against chiropractic.

Eventually, the House of Delegates rescinded its scalding 1966 attack, when it branded chiropractic education as "substandard" and its theory "irrational" and damned that practice as an "unscientific cult". In addition, it dropped its policy of branding as unethical any association of physicians with chiropractors. The change in policy essentially came about because of the legal pressures applied by the suit brought by the Attorney General of the State of New York to recover damages against the AMA, alleging that the Association was engaging in a conspiracy to drive chiropractors out of business, thus driving up their cost of Medicare and Medicaid.

In the end, I would have to say that the almighty dollar won the debate, as Dr. Hotchkiss of the Board of Trustees raised the spectre of endless litigation if the AMA clung to its "unscientific cult" characterization. He pointed out that should New York win this suit, there would be suits in all the other 49 states. And so, the Delegates approved a statement that still declared the AMA knows of no

scientific evidence "to support spinal manipulation as appropriate treatment for ailments" and will continue to warn the public against entrusting diagnosis and treatment to "practitioners who rely on the theory that all disease is caused by misalignment of spinal vertebrae".

I would have to assail this stand as too soft; that honor and integrity are taking a back seat to legal pressures. None of us believes chiropractic is of any value in treating disease, but state and federal government agencies, with their vast monetary resources can grind us into the ground with costly, time consuming lawsuits, caring not one whit about protecting the consumer. The Federal Trade Commission's policy is that quality of care is immaterial as long as it's legal and so we are under the gun from that governmental agency also.

In the end, we may have softened the wording of the AMA stand, but the average physician is still thinking "cult" when he hears of a chiropractor treating an ulcer by manipulating T4.

Sincerely,
D. B. Reaney, M.D., President
South Dakota State Medical Association



M. Frank Petercit, M.D.*

Donald J. Peik, M.D.*



Figure 1

*Radiologists, Medical Xray Center, Sioux Falls, SD.



Figure 2

CASE

The patient is a female in her late 30's with right lower abdominal discomfort for several weeks. Physical examination was negative except for the finding of a vague right lower quadrant mass. Figure 1 shows one of the filled films of a barium enema study. The entire colon fills well but no barium is seen refluxed into the terminal ileum. Figure 2 shows an annular filling defect involving the terminal ileum with suggestive minimal involvement of the cecum. Arrow shows lesion. What is your diagnosis?

DISCUSSION

The differential diagnosis includes regional enteritis (Crohn's Disease), lymphosarcoma, Hodgkin's, abscess and carcinoma. Abscess can usually be excluded by absence of a febrile course or episode

and lack of clinical tenderness. The lymphoblastomas are relatively rare. Carcinoma is possible, but usually involves colon predominantly unless it arises in the small bowel itself which is also rare. Also this patient is quite young. This leaves regional enteritis as statistically most likely. Surgical exploration revealed the lesion to be an adenocarcinoma arising from the cecum, but with unusually great extension into the ileum. This case illustrates the need to establish a definitive diagnosis because the treatment of all the various possibilities in this case would be very different. Colonoscopy or surgical exploration is a necessity if there is any doubt concerning the diagnosis.

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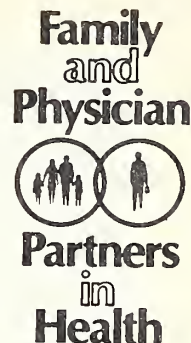
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Batt Memorial Award

Dr. Herbert Saloum, a family physician in Tyn-dall and member of SDAFP Board of Directors, was named the recipient of the first annual Edward J. Batt, M.D. Memorial Award at the USD School of Medicine Spring Awards Banquet.

The award was established in memory of the late Dr. Edward J. Batt and is given to the outstanding DCFM Clinical Faculty member of the year. Dr. Batt was a long-time South Dakota physician and an Associate Professor in the DCFM at the USD School of Medicine. Financial support for the award was made possible through contributions to the USD School of Medicine Endowment Association by fac-

ulty and friends of Dr. Batt.

In making the announcement it was stated, in part, that Dr. Saloum's "...medical student teaching activities represent a broad and deep involvement. He has been a panelist during Family Practice Club activities. He has been involved in teaching in the Introduction to Clinical Medicine course. In addition, he and his partner provide a sophomore preceptorship site, as well as being involved in ongoing clerkship responsibilities in the third year Longitudinal Ambulatory Care Clerkship and fourth year Rural Family Medicine Clerkships. His abilities as a clinical teacher are exemplified by outstanding student evaluations."

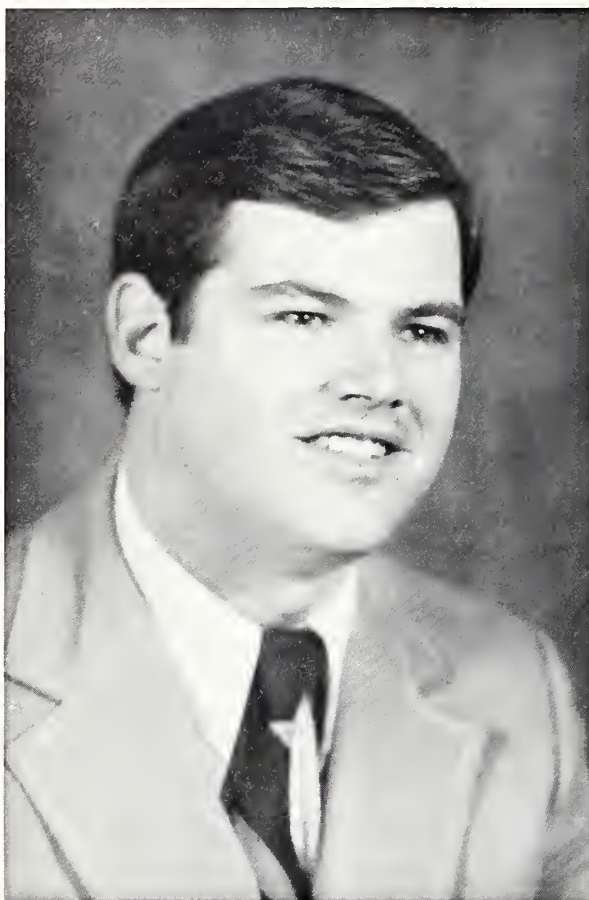
This award will be given annually by the Department of Community and Family Medicine.

Residency Graduate Returns to Program

Dennis Ries, M.D., a 1975 graduate of the Sioux Falls Family Practice Residency, has returned to the United States for a year of sabbatical leave from his missionary post in Zaire, Africa. He and his family are living in Sioux Falls this year while Dr. Ries attends North American Baptist Seminary. He is spending part of his time teaching in the program, and should add an interesting dimension to the resident education program.

Family Practice Residency Reaccredited

Notice has just been received from the Liaison Committee on Graduate Medical Education that the Sioux Falls Family Practice Residency Program has been reaccredited for a three year period. An on-site visitation to the program was made by the Residency Review Committee for Family Practice in April of this year leading up to the review and accreditation process. A yearly progress letter to the LCGME has been requested.



IF YOU'RE MAD, CALL PETER

As Vice President of Public and Professional Relations, one of Peter Galindo's responsibilities is to protect your rights with regard to South Dakota Blue Shield and the various programs it administers.

We believe that you have certain rights such as the right to speed, efficiency and accuracy in claims processing. The right to courteous service. The right to fair treatment. The right to a satisfactory explanation if a claim is turned down, or if the benefit allowance is substantially reduced.

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He will investigate the matter thoroughly. If the problem is a result of our shortcomings, it is Peter's job to see that steps are taken to prevent it from happening again.

Peter isn't here to answer routine questions on claim filing, we have a Field Representative for that. Peter is here for when you get "steamed up" and normal channels don't work. If that happens, call Peter Galindo at 336-1976.

All of us at Blue Shield are doing our best to see that you never have to call Peter.

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Sioux Falls, South Dakota 57104
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SD

This Is Your Medical Association

Jorge E. Sanmartin, M.D., Rapid City, and **Don Kough, M.D.**, Sioux Falls, were elected to the board of directors of the American Heart Association, Dakota Affiliate; each to serve a two-year term.

* * * *

Rapid City Medical Center has announced the appointment of **Elmo J. Rosario, M.D.** to the department of internal medicine. Dr. Rosario graduated from Baylor College in 1973. He completed his internship, residency and a pulmonary fellowship at Creighton Affiliated Hospitals in Omaha, Nebraska.

* * * *

Charles E. Hollerman, M.D. has been named dean of the University of South Dakota School of Medicine and vice president of health affairs. Dr. Hollerman has been acting dean of the school since February. Dr. Hollerman resides in Yankton where he is associated with the medical staff at Sacred Heart Hospital. He earned his medical degree at Cornell University Medical College in 1955 and completed his internship at York County Hospital in Pennsylvania. He joined the School of Medicine faculty in 1975 as a professor of pediatrics. He was named assistant dean for clinical sciences curriculum in 1976 and served as acting executive dean for five months before accepting permanent responsibility in that position in July of 1977. Dr. Hollerman assumed the new position August 1.

Five Sioux Falls people have been named to Alpha Omega Alpha the only national honor medical society in the world. They are **Judith Gravdal** and **Wendell Hoffman**, seniors at USD School of Medicine; **David R. Johnson**, a third-year medical student, and **Robert Van Demark M.D.**, and **John Barlow, M.D.**, both practicing physicians in Sioux Falls. There are now 72 Alpha Omega Alpha members practicing in South Dakota.

* * * *

Stanley J. Gerrick, M.D. has joined the staff at Day County Medical Center at Webster. Dr. Gerrick is a graduate of Wayne State University, Detroit, Michigan. He recently completed his five year residency as a general surgeon at Wayne State University.

**YOUR
CONTRIBUTION
IS NEEDED
TO THE
SOUTH DAKOTA
MEDICAL SCHOOL
ENDOWMENT
FUND**

Wm. R. Taylor, M.D., Aberdeen, has been named chairman of the Aberdeen United Way Campaign.

* * * *

Wm. G. M. Huet, M.D. of Huron and **Hubert G. Werthmann M.D.** of Pierre were named Fellows of the American College of Radiology in recognition for distinguished medical achievements. The College awarded their certificates of Fellowship during its annual meeting and convocation in Chicago.

* * * *

The appointment of **James J. Pavlovich, M.D.** to the pediatric department has been announced by the Rapid City Medical Center. Dr. Pavlovich graduated from Creighton University Medical School in 1973 and completed his internship at St. Joseph Hospital in Omaha. He recently completed his pediatric residency at University of Nebraska prior to coming to Rapid City.

* * * *

H. Stephen Williams, M.D. recently opened his office for the practice of pediatrics in Mitchell. Dr. Williams had a pediatrics practice in Los Angeles, California prior to moving to Mitchell. He graduated from Johns Hopkins University School of Medicine in 1970. He served a year of internship and a year of pediatric residency at Johns Hopkins and a year of residency at Los Angeles County Harbor General Hospital.

(continued)

Charles L. Paxson, M.D., neonatologist at Central Plains Clinic, Sioux Falls, attended the 47th annual meeting of the American Bar Association, held at the Sheraton-Dallas Hotel in Dallas, Texas. Dr. Paxson spoke to the Section of Negligence and Compensation Law which is part of the "Law and Medicine Committee" of the American Bar Association. He spoke at a symposium entitled "Legal Aspects of the Provision and Withdrawal of Medical Care to the Defective Newborn Infant".

* * * *

Phillip E. Hoffsten, M.D. has

joined the staff of the Medical Associates Clinic in Pierre specializing in internal medicine. Dr. Hoffsten is a graduate of Washington University, St. Louis, Missouri. He served his internship at Vanderbilt University Hospital, Nashville, Tennessee and his residency at Barnes Hospital, St. Louis. Prior to moving to Pierre, Dr. Hoffsten was assistant professor in the department of medicine at Washington University for six years.

* * * *

Mike Haley, M.D. started a surgical practice in Mitchell recent-

ly. Dr. Haley, a graduate of the University of South Dakota School of Medicine, has been practicing in Yankton until his move to Mitchell.

* * * *

A Sioux Falls physician has been named to work on two infant health programs. **Stanley N. Graven, M.D.**, Professor of Pediatrics and Adolescent Medicine, USD Medical School, will direct a 13-month Rural Infant Care Program and also be involved in The Work Group on Pregnancy and Infant Care.



"When I speak out for the rights of retarded people, I know what I'm talking about, because I'm one of them."

Michael is part of the Association for Children with Retarded Mental Development (A.C.R.M.D.), an organization that fights discrimination against retarded people. He has been their spokesman at many meetings.

Michael has managed to get jobs for a number of retarded people by talking directly to politicians. They call the A.C.R.M.D. and say that a young man from your organization spoke up and we're interested in hiring someone.

Michael is one of the first retarded people to work full time for the Federal Government. After he was on the job a few years they gave him a special citation for improving the efficiency of the office.

"I had the idea of using a less expensive paper for the office copying machines. It saves the government thousands of dollars a year."

Michael says that the determination of the handicapped to prove themselves capable of doing a job well and thoroughly is the best argument for hiring them.

President's Committee on
Employment of the Handicapped
Washington, D.C. 20210
The School of Visual Arts Public Advertising System

...in the functional bowel/irritable bowel syndrome*

Bentyl[®]

(dicyclomine hydrochloride USP)

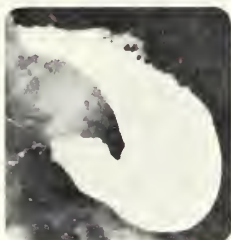
10 mg. capsules, 20 mg. tablets,
10 mg./5 ml. syrup, 10 mg./ml. injection

helps control abnormal motor activity
with minimal anticholinergic side effects†

Demonstrated smooth muscle relaxant activity.

In this double-blind study, twenty patients having G.I. series and exhibiting spasm were randomly selected to receive either 2 cc. of Bentyl or sodium chloride intramuscularly. Ten minutes after the injection another radiograph was taken . . .

. . . Bentyl produced definite relaxation in 8 of 10 patients. The sodium chloride produced relaxation in only 3 of 10. No side effects occurred in either group of patients.



Pylorospasm has almost totally blocked passage of barium meal.



Barium meal beginning to pass 10 minutes after intramuscular injection of 20 mg. Bentyl.

"The correlation of spasm relief and drug given was excellent."

*This drug has been classified "probably" effective in treating functional bowel/irritable bowel syndrome.

†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:

King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

Merrell

Bentyl[®]**(dicyclomine hydrochloride USP)**

Capsules, Tablets, Syrup, Injection

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

INDICATIONS

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the following indications as "probably" effective:

For the treatment of functional bowel/irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

THESE FUNCTIONAL DISORDERS ARE OFTEN RELIEVED BY VARYING COMBINATIONS OF SEDATIVE, REASSURANCE, PHYSICIAN INTEREST, AMELIORATION OF ENVIRONMENTAL FACTORS.

For use in the treatment of infant colic (syrup).

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloroduodenal stenosis); paralytic ileus, intestinal atony of the elderly or debilitated patient; unstable cardiovascular status in acute hemorrhage; severe ulcerative colitis; toxic megacolon complicating ulcerative colitis; myasthenia gravis. **WARNINGS:** In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. **PRECAUTIONS:** Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy. Use with caution in patients with: Autonomic neuropathy. Hepatic or renal disease. Ulcerative colitis. Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon. Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension. Hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.

Do not rely on the use of the drug in the presence of complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. **ADVERSE REACTIONS:** Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia; urinary hesitancy and retention; blurred vision and tachycardia; palpitations; mydriasis; cycloplegia; increased ocular tension; loss of taste; headache; nervousness; drowsiness; weakness; dizziness; insomnia; nausea; vomiting; impotence; suppression of lactation; constipation; bloated feeling; severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating. With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. **DOSE AND ADMINISTRATION:** Dose must be adjusted to individual patient's needs.

Usual Dosage: Bentyl 10 mg. capsule and syrup: *Adults:* 1 or 2 capsules or teaspoonfuls syrup three or four times daily. *Children:* 1 capsule or teaspoonful syrup three or four times daily. *Infants:* ½ teaspoonful syrup three or four times daily (May be diluted with equal volume of water.) Bentyl 20 mg.: *Adults:* 1 tablet three or four times daily. Bentyl Injection: *Adults:* 2 ml. (20 mg.) every four to six hours intramuscularly only. **NOT FOR INTRAVENOUS USE.** **MANAGEMENT OF OVERDOSE:** The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal. Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested. If indicated, parenteral cholinergic agents such as Urecholine[™] (bethanechol chloride USP) should be used.

Product Information as of October, 1978.

Injectable dosage forms manufactured by CONNAUGHT LABORATORIES, INC., Swifwater, Pennsylvania 18370 or TAYLOR PHARMACAL COMPANY, Decatur, Illinois 62525 for MERRELL-NATIONAL LABORATORIES, Division of Richardson-Merrell Inc., Cincinnati, Ohio 45215, U.S.A.

S D

Laboratory Aids

Sponsored by the South Dakota Society of Pathologists

Thin Needle Aspiration Biopsy

Thin needle (skinny needle) aspiration biopsy is a diagnostic method that has recently gained increasing acceptance in this country.

This technique is not new; the first published reports were from Memorial Hospital in New York City in 1926. European centers have been utilizing this method extensively since the 1950's.

Thin needle aspiration biopsy affords us a means of making a definitive tissue diagnosis in an almost nontraumatic manner in lieu of open biopsy or tissue core biopsy.

The technique involves placing a small gauge needle (21 to 25 gauge) within a lesion and then moving the needle back and forth through the tissue while applying negative pressure. A very scant amount of tissue fluid is then obtained. This material, which may not even appear in the syringe, is then placed on slides and smears are made. These smears may then be stained in a variety of ways so the pathologist can look for cell morphology as well as for acid fast and fungal organisms. Our technique calls for immediate fixation but techniques used by others may not require this.

The drawback to this procedure is that the pathologist is looking at individual cells and this requires meticulous preparation of the smears. For this reason, we like to be present at the time the aspirate is obtained. From a clinical point of view there has been a fear of spreading tumor along the needle tract. Although there has been a rare case report of this phenomenon, it has not been a problem in the European experience.

The major advantage of this procedure is that it enables us to obtain a definitive diagnosis in lesions of the intrathoracic, thyroid, lymph nodes, retroperitoneal and intra-abdominal areas without open biopsy. For patients with benign tumors, infectious processes or disseminated malignancy, this technique will often make major surgery unnecessary.

We feel that the needle aspiration biopsy with guidance by fluoroscopy, ultrasound and CAT scanners can lead to more rapid diagnosis, shorter hospital stays and fewer open biopsies in selected cases when a tissue diagnosis is needed.

Lawrence Seidenstein, M.D.

Pathologist

Merrell

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SOUTH DAKOTA

THE ALUMNI ASSOCIATION

of the

SOUTH DAKOTA SCHOOL OF MEDICINE

has been established by the South Dakota Medical School Endowment Association. Among other activities, the Alumni Association serves as a source of information for graduates and assists in the organization of class reunions.

As of 1977 the South Dakota School of Medicine is a four-year degree granting school, and through the Alumni Association the school, and past and present students will be better served.

Contributions may be sent to:

**Alumni Association
South Dakota Medical School
Endowment Association
608 West Avenue, North
Sioux Falls, South Dakota 57104**

SD *Future Meetings*

October

Principles of Colon and Rectal Surgery, Mayo Aud., U. of Minn., Minneapolis, MN, Oct. 31-Nov. 3. Fee: \$235. 26 hrs. AMA Category 1 credits. Contact: CME Off., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

November

Plastic Surgery, Towsley Ctr. for CME, U. of Mich. Med. School, Ann Arbor, MI, Nov. 1-3. Contact: Off. of CME, U. of Mich. Med. School, Towsley Center for CME, Ann Arbor, MI 48109. Phone: (313) 764-2287.

45th Annual Scientific Assembly of the American College of Chest Physicians, Hyatt Regency Hotel and the Albert Thomas Convention Hall, Houston, Tex., Nov. 4-8. 30 hrs. AMA Category 1 credits. Contact: Dale E. Braddy, Dir. of Ed., ACCP, 911 Busse Highway, Park Ridge, IL 60068.

Refraction for the Non-Ophthalmologist, Unit B/C, U. of Minn., Minneapolis, MN, Nov. 7-9. 24 hrs. AMA Category 1 credits. Contact: CME Off., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Tumors of the Breast: Morphologic and Clinical Aspects, Mayo Aud., U. of Minn., Minneapolis, MN, Nov. 8-9. 13 hrs. AMA Category 1 credits. Contact: CME Off., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Perinatology for Family Physicians, St. Paul Ramsey Med. Ctr., St. Paul, MN, Nov. 8-9. 14 hrs. AMA Category 1 credits. Contact: CME Off., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Physician Compensation and Contracting, Sheraton Plaza, Chicago, IL, Nov. 8-10. Contact: Aspen Systems Corp., 20010 Century Blvd., Germantown, MD 20767. Phone: (301) 428-0700.

Accent on Youth, Hilton Inn, Albuquerque, NM, Nov. 8-10. 6 hrs. AMA Category 1 credits. Contact: Am. Medical Women's Assoc., 1740 Broadway, New York, NY 10019. Phone: (212) 586-8683.

Medical Care and Health Services in Correctional Institutions, Radisson Chicago Hotel, Chicago, IL, Nov. 9-10. Fee: \$60. 14 hrs. AMA Category 1 credits. Contact: AMA Correctional Prog., AMA, 535 N. Dearborn St., Chicago, IL 60610.

Sexual Attitude Reassessment Seminar, Research East Bldg., U. of Minn., Minneapolis, MN, Nov. 9-10. Fee: \$150. 16 hrs. AMA Category 1 credits. Contact: CME Off., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Current Problems as the Interface of Law and Medicine, Ambassador West Hotel, Chicago, IL, Nov. 10. 7 hrs. AMA Category 1 credits. Contact: Am. College of Legal Medicine, 875 N. Michigan Ave., Ste. 3342, Chicago, IL 60611.

Otorhinolaryngology, Towsley Ctr. for CME, U. of Mich. Med. School, Ann Arbor, MI, Nov. 16-17. Contact: Off. of CME, U. of Mich. Med. School, Towsley Ctr. for CME, Ann Arbor, MI 48109. Phone: (313) 764-2287.

Time-Management-Money: A Briefing and Update for Office Practice, Sheraton-Ritz Hotel, Minneapolis, MN, Nov. 16-17. Fee: \$100. 10 hrs. AMA Category 1 credits. Contact: CME Off., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Workshop on Practice Productivity, Desert Inn, Las Vegas, NV, Nov. 16-17. Contact: Am. College of Obstetricians and Gynecologists, One E. Wacker Dr., Suite 2700, Chicago, IL 60601. Phone: (800) 421-6512.

Mayo Foundation Outreach Seminar, McKennan Hosp. Aud., Sioux Falls, SD, Sept. 28-29. AMA Category 1 credits and AAFP credits. Contact: Off. of Med. Ed., McKennan Hosp., 800 E. 21st St., Sioux Falls, SD 57101.

Your Cancer Patients—Maintaining Quality of Life, Bethesda Lutheran Hosp., St. Paul, MN, Nov. 29-30. 14 hrs. AMA Category 1 credits. Contact: CME Off., Box 293 Mayo

Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Patient Care Evaluation Techniques for the 80's, Los Angeles, CA, Nov. 29-30. Fee: \$195. 14 hrs. AMA Category 1 credits. Contact: InterQual, 740 N. Rush, Chicago, IL 60611. Phone: (312) 751-2327.

The Supercourse V, Postgraduate Program on Lung Disease, Hyatt Regency Hotel, New Orleans, LA, Nov. 27-Dec. 1, AAFP and AMA Category 1 credit hrs. Fee: \$225. Contact: John B. Bobear, M.D., Supercourse V Chairman, American Lung Assoc. of LA, Inc., Suite 500, 33 St. Charles Ave., New Orleans, LA 70130.

December

Cardiology Today, U. of Iowa Hosp., Iowa City, IA, Dec. 3-6. Fee: \$275. 30 hrs. AAFP & AMA Category 1 credits. Contact: Carl W. White, M.D., Cardiovascular Div., U. of Iowa Hosp., Iowa City, IA 52242.

The Fourth Southeastern Conference on Alcohol and Drug Abuse, Atlanta, GA, Dec. 5-9. 27 hrs. Category 1 credits. Contact: Conway Hunter, M.D., Peachford Hosp., 215 Peachford Road, NE, Atlanta, GA 30338. Phone: (404) 455-3200.

Care of Burned Patient, Towsley Ctr. for CME, U. of Mich., Ann Arbor, MI, Dec. 7. Contact: Off. of CME, U. of Mich. Med. School, Towsley Ctr. for CME, Ann Arbor, MI 48109. Phone: (313) 764-2287.

Clinical Nutrition for the Practicing Physician, Sheraton-Ritz Hotel, Minneapolis, MN, Dec. 7-8. 10 hrs. AMA Category 1 credits. Contact: CME Off., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Continuing Pathology, Towsley Ctr. for CME, U. of Mich., Ann Arbor, MI, Dec. 11. Contact: Off. of CME, U. of Mich. Med. School, Towsley Ctr. for CME, Ann Arbor, MI 48109. Phone: (313) 764-2287.

Workshop on Coronary Heart Disease, Spring Hill Ctr., Wayzata, MN, Dec. 12-14. 17 hrs. AMA Category 1 credits. Contact: CME Off., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

SOUTH DAKOTA JOURNAL OF MEDICINE

Published Monthly by the S.D. State Medical Assn.

Volume XXXII November 1979 Number 11

The Physician and the Hyperactive Child

Clinicopathological Conference

Fifty-Two Year Old Man with Urethral Bleeding

Proteinuria, and Abnormal Serum Proteins

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fenoprofen calcium

300-mg.* Pulvules[®] and 600-mg.* Tablets



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*Additional information available to the profession
on request.*

*Present as 345.9 mg. and 691.8 mg. of the calcium salt of fenoprofen dihydrate equivalent to 300 mg. and 600 mg. fenoprofen respectively.

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72.



A character all its own.



Valium (diazepam/Roche) is a benzodiazepine with a character all its own.

Pharmacologically, it is a potent skeletal muscle relaxant and anticonvulsant (in adjunctive use), as well as an antianxiety agent. Pharmacokinetically, only Valium provides active *diazepam* as well as the active metabolites 3-hydroxydiazepam, desmethyldiazepam and oxazepam.

But the individual character of Valium is even more apparent clinically than pharmacokinetically. And far more significant. That's because of the patient response obtained with Valium. A response which brings a calmer frame of mind. A response which has a pronounced effect on the somatic symptoms of anxiety, particularly muscular tension. A response which helps the patient feel more like himself again because of the way Valium reduces the overwhelming symptoms of anxiety and psychic tension.

Another important aspect of the clinical character of Valium is safety. Though drowsiness, ataxia and fatigue are possible, these and more serious side effects are rarely a problem. Of course, as with all CNS-acting drugs, patients taking Valium should be cautioned against driving, operating dangerous machinery or the simultaneous ingestion of alcohol.

Unquestionably, many psychotherapeutic agents, including other benzodiazepines, have antianxiety effects. But one fact remains: you get a certain kind of patient response with Valium. It's a response you want. A response you know. A response you trust as part of your overall management of anxiety and psychic tension.

Valium®^{IV}
diazepam/Roche
2-mg, 5-mg, 10-mg scored tablets
a prudent choice in psychic
tension and anxiety

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.



Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

SOUTH DAKOTA JOURNAL OF MEDICINE

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SCIENTIFIC ARTICLES

- 7 The Physician and the Hyperactive Child**
Carl N. Rutt, M.D.
- 25 Clinicopathological Conference**
Fifty-Two Year Old Man with Urethral Bleeding
Proteinuria, and Abnormal Serum Proteins
Milton C. Zadina, M.D.
John F. Barlow, M.D.

FEATURES

- 12 Letters to the Editor**
- 19 President's Page**
- 21 Editorial**
- 32 Laboratory Aids**
- 37 South Dakota AFP Chapter News**
- 38 Future Meetings**

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The Physician and the Hyperactive Child

Carl N. Rutt, M.D.*

ABSTRACT

Hyperactive children, usually boys, are frequently referred to family physicians for diagnosis, evaluation and treatment. Diagnosis includes hyperactive, impulsive, and excitable behavior, short attention span and distractibility, learning disabilities, and symptoms which are present in many situations. Possible etiologies include organic brain dysfunction, developmental hyperactivity, genetic predispositions, emotional disturbances, and combinations of the above. Although hyperactivity may diminish as the child grows older, active intervention is usually re-

quired, since morbidity includes school failure, poor peer relationships, and delinquency. A comprehensive treatment plan is essential, and includes medication, behavior modification, counseling, and specific educational intervention. Medication should be used to treat specific target symptoms, and monitoring of progress is possible with the Conner's Teacher Rating Scale. The role of food additives and sugar remains controversial, and claims regarding effectiveness of special diets have not been subjected to adequate scientific scrutiny.

INTRODUCTION

Few childhood disorders create such medical controversy, or family upheavals, as hyperactivity. But it has been more than 100 years ago that the original description of hyperactivity was provided by Heinrich Hoffman, a German physician.

A multitude of terms have been applied to this condition, ranging from "hyperactive" and "hyperkinetic" to pejorative terms such as "minimal brain damage" or "minimal brain dysfunction". There is also confusion regarding learning disabilities and how they are related to the hyperactive child syndrome. In this review, for simplicity, the term hyperactive will be used.

Family physicians commonly evaluate hyperactive children after referral by school personnel. However, most hyperactive children have had signs of behavior disturbance earlier in childhood, often by two years of age.¹ Parents may sense their child has a problem, but may not understand its significance, or know what to do about it. Why 90% of hyperactive children are boys is unknown.

DIAGNOSIS

A group of signs and symptoms make the diagnosis readily apparent in most hyperactive children. Physicians should make the diagnosis using not only their own careful observations, but also reports from parents and teachers, since a single interview may be equivocal or misleading.

a) Hyperactive behavior. The child is physically over-active, fidgets, squirms, and makes random, purposeless movements. Commonly, he walks about the room, or may dart, lunge, or run. He acts as if he were driven by an inner motor, and may talk incessantly. Parents complain he is always on the go and "gets into things."

b) Short attention span. Most hyperactive children have difficulty sticking to things. Their attention may be focused for a few seconds or a minute, but they rarely finish a project, or may have difficulty watching a complete TV program. In addition, other stimuli (noises, talking) may distract the child and he abandons the prior activity.

c) Impulsive and excitable. Hyperactive children typically do things without anticipating the consequences of their behavior. They get an urge to do something and they act on the impulse. This leads

*Assistant Professor, Departments of Psychiatry and Pediatrics and Adolescent Medicine, USD School of Medicine, 2501 W. 22nd St., Sioux Falls, SD.

them to take excessive risks, break items, and become reckless and destructive. Parents complain they get wound up too easily, and as a result, may withdraw from their child. "He gets wound up so easily, and then can't stop—I don't even feel like playing with him." Many are unpredictable and respond poorly to discipline. The impulsive behavior disturbs their peer relationships.

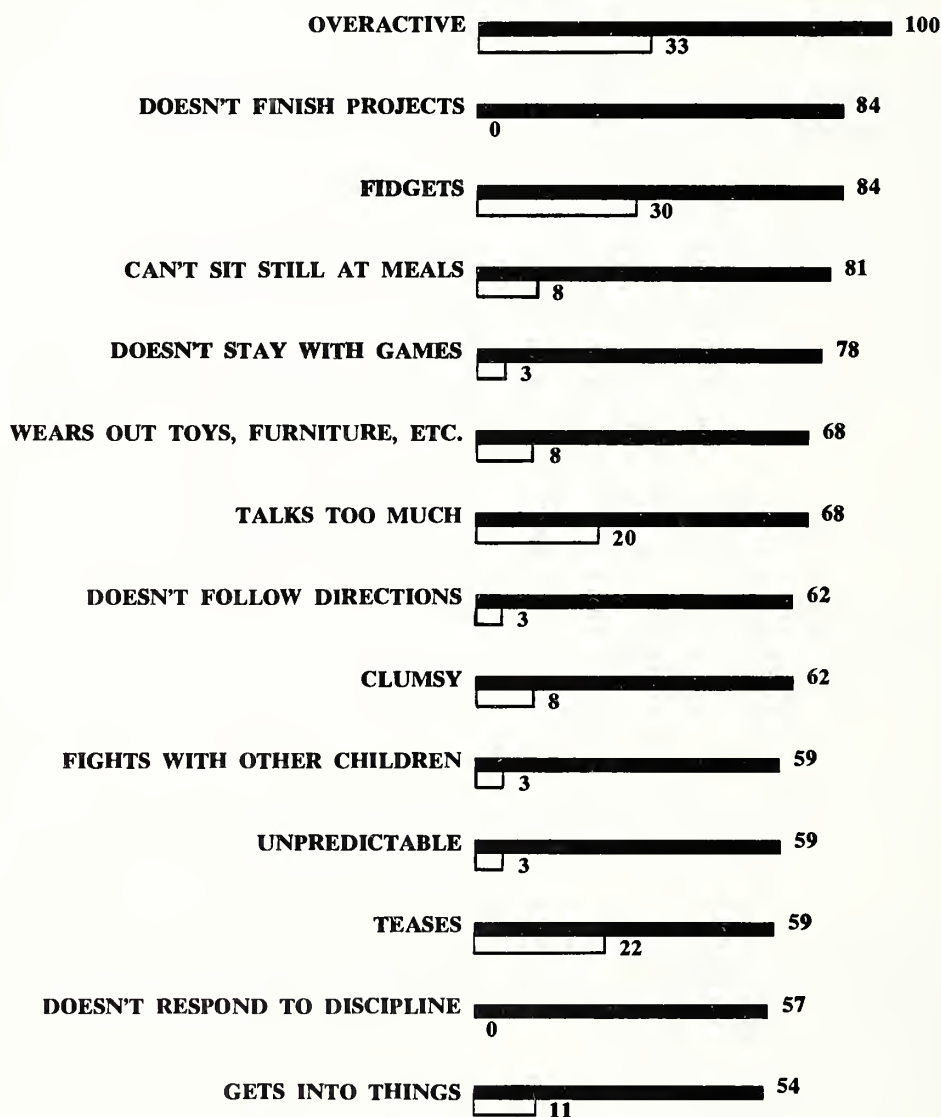
d) Many hyperactive children develop learning disabilities as a result of impaired attention span. This frequently produces a low self-concept, and the

child feels he is "dumb". He may then find inappropriate ways to assert himself and gain attention—for example, by being the class clown or bully.

e) Symptoms are present in many situations (home, school, public places). If difficulty only occurs in one setting, a restructuring of that environment may be necessary, e.g. clearer limits, and the child is probably not truly hyperactive.

Stewart compared 37 hyperactive schoolchildren (32 boys and 5 girls) ages 5 to 11, with normal children, and results are summarized in Table 1.

TABLE I
Common Symptoms of Hyperactivity
(derived from Mark A. Stewart)¹
Percent of Children showing each symptom



KEY: ■ Hyperactive child
□ Normal child

ETIOLOGY

a) Brain damage. Many years ago, Strauss and Lehtinen² noted that some children with definite brain damage were more hyperactive, distractible and impulsive than non brain-injured children. This led to circular reasoning and the view that children showing these symptoms must therefore be brain damaged. As a result, this led to the labels "minimal brain damage" or "minimal brain dysfunction". Unfortunately, such terms convey to parents the specter that their child actually has brain damage. In reality, this is false, since studies of children with short attention span and impulsive, excitable behavior show that most have no definite EEG or brain scan abnormalities.³ For example, Satterfield found that only 35-50% of hyperactive children have EEG abnormalities, with increased slow waves the most common finding.⁴ For this reason, the less emotionally-laden terms "hyperactive" or "hyperkinetic" are most desirable. (In a few years, the diagnosis "attention deficit disorder with/without hyperactivity" might replace these other terms.)

b) Developmental hyperactivity. Young children typically are more active, having more gross body movements of arms and legs, than older children or adults. In this sense, hyperactivity may be a "normal" component of development.

However, in a group of 5-year-olds, for example, a range of activity level would be apparent, ranging from fairly quiet, placid children to average children, to active, flighty, fidgety children who talk incessantly. Is the hyperactive child simply at the extreme end of the activity level scale? Possibly. However, recent neurophysiologic studies suggest that a subgroup of hyperactive children actually have "low central nervous system arousal."^{3,5}

c) Genetic. Family studies show that about 10% of the biologic parents of hyperkinetic children were hyperkinetic themselves as children.⁶ Additional studies of hyperkinetic children who were placed in adoptive homes soon after birth found a high rate of alcoholism, antisocial behavior, and hysteria in biologic parents. The hyperactive child syndrome is more common among brothers of hyperactive children than among brothers of controls (26% vs. 9%).⁷ Such studies suggest a genetic etiology or other possible causes.

d) Emotional disturbance. Poorly supervised, neglected children may show overactivity. Some children who appear hyperactive are actually anxious, troubled by worries about home or school problems. However, this can be determined by age of onset (later than the true hyperactive), or by identifying specific stresses the child is responding to. In addition, hyperactive children are sometimes thought to

represent a "depressive equivalent".

e) In general, multiple factors are probably responsible for hyperactive behavior.

CLINICAL COURSE AND OUTCOME

As the child grows older, the hyperactive behavior tends to diminish and attention span often increases. However, this fact has led to over-optimism. There is significant concern now about the toll produced by this disorder, e.g. children who drop out of high school, develop delinquent behavior patterns, and fail to develop friendships. Physicians may advise to "wait and see if he'll grow out of it". This is risky and should be avoided. Mendelson found that over half of adolescent hyperactives had problems with fighting, stealing, and destructive behavior, and one-third had threatened to kill a parent. In school, over half had failed one or more grades, 18% had failed two or more grades.

TREATMENT

In general, a comprehensive treatment plan is preferred to simply using one form of treatment, and a cooperative working alliance with parents and teachers is essential.

a) Medication. A most remarkable finding in pharmacology was the observation by Bradley in 1937 that benzedrine calmed children who were hyperactive.⁹ This led to the concept of the "paradoxical effect"—that a stimulant drug calms children. For some children, this effect may not be paradoxical at all—the calming effect may be a logical result of stimulating inhibitory centers, or raising arousal level. In recent years, Ritalin, Dexedrine, and Cylert have been used successfully with three-quarters of hyperactive children.

With some children, the response to stimulant drugs is dramatic, sudden, and very gratifying. The aggressive, impulsive, school child who requires constant attention by the teacher may, by midmorning, after the first dose of medication, suddenly become a polite, attentive pupil. Many children will not show such a profound or immediate change, having built up patterns of behavior, and being used to acting out to get attention. Some will show no response at all. Safer reports that when "stimulants are given in an adequate dose to obviously hyperactive children, 35-50% show dramatic benefit, 30-40% show moderate benefit, and 15-20% show no benefit."¹⁰ Frequently the child improves in several areas—academically, socially, and emotionally.

Ritalin is commonly started at 5 or 10 mg. before breakfast and lunch, and increased by 5 to 10 mg. doses every 2 or 3 days. Some children whose prob-

blems are mainly expressed at school can be managed by twice a day meds, five days a week. Others who are equally overactive at home and at school need meds before breakfast, lunch, and about 4:00 p.m. seven days a week.

Many children can be managed on 20 to 30 mg. per day; others might receive adequate response at 40, 60, 80 mg. Occasional reports of 200 mg. per day have appeared. In any event, the dose is titrated to the child's needs, starting small and increasing as necessary.

Dexedrine Spansule capsules (5, 10, 15 mg.) allow once a day dosage. Dexedrine tablets (5 mg.) are given two or three times a day. Cylert is given once a day as an 18.75 mg., 37.5 mg., or 75 mg. tablet.

Occasionally, Mellaril 25 to 50 mg. two to three times a day provides benefit for children not helped by stimulants. However, Mellaril may overly sedate the child and decrease learning or alertness in school. Antidepressants (Elavil, Tofranil) have also surprisingly helped some children.

Medication is used to treat specific target symptoms, and it is very essential to monitor progress. Teacher rating scales, devised by Conners, provide accurate baseline and follow-up assessment of the hyperactive child's behavior in school.¹¹

The abbreviated, 10-item scale (See Table II) is useful for the physician to monitor. Any child scoring 15 or more would be clearly hyperactive. This should be filled out by the teacher prior to beginning medicine, then at weekly to biweekly to monthly intervals as treatment progresses.

Trials off medication are desirable, perhaps during school days, or in the summer. A sudden or gradual relapse calls for re-instituting the medication. Long term side effects include increased heart rate and minor growth suppression. However, after medication is decreased, there may be a compensatory rebound in height and weight.¹² There is no evidence that drug abuse tends to follow stimulant use, although this has not been answered for sure. Other side effects include appetite suppression, dazed appearance, headaches, stomach aches, and moodiness.

Children often do not like the stimulants, even when deriving major benefit from them. Children need to be educated that the pills are an aid to help them gain control over their impulses, but that bad behavior can't be excused because they forgot their pill.

Parents' and teachers' concerns about "drugging" children argue for careful selection of which children

TABLE II
TEACHER RATING SCALE
(Abridged from C.K. Conners)¹¹

	0 Not at all	1 Just a Little	2 Pretty Much	3 Very Much
1. Restless and overactive	_____	_____	_____	_____
2. Excitable and impulsive	_____	_____	_____	_____
3. Disturbs other children	_____	_____	_____	_____
4. Fails to finish things he starts, short attention span.	_____	_____	_____	_____
5. Constantly fidgeting.	_____	_____	_____	_____
6. Inattentive, easily distracted.	_____	_____	_____	_____
7. Demands must be met immediately, easily frustrated.	_____	_____	_____	_____
8. Cries often and easily.	_____	_____	_____	_____
9. Mood changes often and drastically.	_____	_____	_____	_____
10. Temper outbursts, explosive and unpredictable behavior.	_____	_____	_____	_____
Note: Total score is obtained by adding numbers in each column.				

truly need medication. It is one important aid in a comprehensive management program, and not a panacea.

b) Behavior modification offers a powerful tool for dealing with specific, maladaptive behaviors of many children. For example, parents and teachers can set up a reinforcement schedule for the child who does not stick to a school assignment or a project at home.

The problem area is precisely defined—for example, how long is the child's attention span. Verbal rewards (praise) and tangible rewards (privileges) can be awarded the child for gradual increase in length of time he can attend to a task. Time out (on chair or in bedroom) can squelch destructive interactions. Forming daily routines at home and school can aid the child in forming inner habits and structure. Attention is itself reinforcing to children—unfortunately parents often pay more attention to their children when they are misbehaving. Many inappropriate behaviors can be ignored by parents, causing them to disappear.

c) Counseling techniques—ventilation of attitudes within the family of a hyperactive child can be highly therapeutic. Frequently, parents feel hostility and resentment toward their overactive child, who on the other hand, feels shame and senses his parents' displeasure. Working to make the parent-child interaction positive pays off in increased maturation of both parents, child and siblings. Several effective, readable programmed learning books for parents include **Living With Children** by Patterson and Guillion,¹³ and **Families** by Patterson.¹⁴ Physicians might have these available in their offices or loan them out to patients.

d) Educational intervention is usually necessary, since most hyperactive children perform poorly in some or many school subjects. Individualized instruction, reducing distractions, firm limits, and immediate feedback may boost the child's learning. Ideally the child can remain in the regular classroom, but if he is too far behind academically, holding him back a grade or placing him in special education can allow the child to start having successful experiences again. A newer concept is that of the resource room, in which the child spends perhaps 1-3 hours a day in a special room, and is taught by a special education teacher in an effort to give remedial help in specific areas of deficit.

e) Other treatments, of a more controversial nature, include the Feingold K-P Diet,¹⁵ eliminating sugars and food additives, and megavitamin therapy.

These methods deserve further study; some claims appear exaggerated, and haven't been subjected to adequate scrutiny. Improvement may represent

placebo responses—for example, the special attention the child receives during the dietary regimen may change family interaction patterns enough to account for the therapeutic change.

Conners, in a preliminary, double blind assessment of the K.P. Diet, using 15 children, suggested "that a diet free of most natural salicylics, artificial flavors, and artificial colors reduces the perceived hyperactivity of some children", but emphasized this should be interpreted with caution.¹⁶

SUMMARY

Hyperactivity is a complex disorder, and frequently creates major childhood and familial disability. Extreme positions should be avoided, such as denying its existence (see **The Myth of the Hyperactive Child**¹⁷), as should the opposite tendency to treat every hyperactive child with medication.

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Letters To The Editor

Scholarship

As a recent recipient of the Memorial Merit Scholarship, I would like to convey my gratitude to all members of the South Dakota Chapter of the American Academy of Family Physicians. I intend to utilize this award in educational pursuit and look forward to family practice in South Dakota

Sincerely,
Craig K. Hansen, M.D.

Clarification of Procedure

I recently presented a discussion on office procedures at the Family Practice Seminar in Spearfish. I would like to reemphasize one point in regard to office breast biopsies in that the tissue should be frozen with dry ice or a cryostat as soon as possible and certainly within a half hour of removal of the tissue. The best procedure to follow in preparation of this tissue is to consult with your pathologist and ask him specifically how he would like to have you handle the tissue. Estrogen receptor analysis of carcinoma of the breast is an extremely important test and certainly should be part of the permanent record of the patient.

Sincerely,
R. R. Lawrence, M.D., F.A.C.S.


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Usage in pregnancy Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. Therefore, PERCOCET®-5 should not be used in pregnant women unless, in the judgment of the physician, the potential benefits outweigh the possible hazards.

Usage in children PERCOCET®-5 should not be administered to children.

PRECAUTIONS **Head injury and increased intracranial pressure** The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a pre-existing increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.

Acute abdominal conditions The administration of PERCOCET®-5 or other narcotics may obscure the diagnosis or clinical course in patients with acute abdominal conditions.

Special risk patients PERCOCET®-5 should be given with caution to certain patients such as the elderly or debilitated, and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, and prostatic hypertrophy or urethral stricture.

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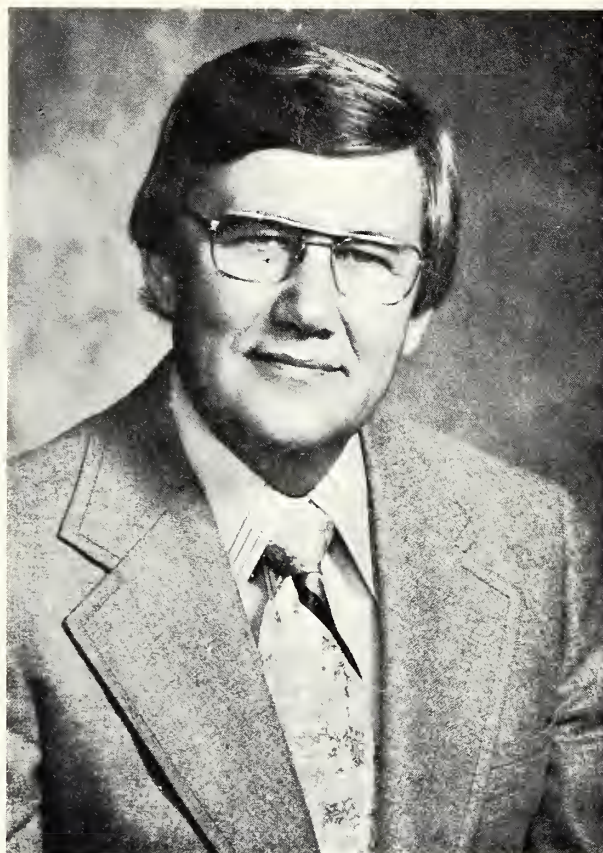
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President's Page

Not too long ago, the President of the United States reminded us that health care costs in this country had increased 1,000% from 1950 to 1977. These figures we probably cannot refute. However, the President nor any of his staff pointed out that the medicine that was being bought in 1950 does not resemble the 1977, 1978 or the present day model whatsoever.

In fact, if in 1950 you had a heart condition—a bad valve or bad coronaries—you could take diuretics and digitalis with some relief. When those medicines no longer worked, you got short of breath and you probably died. In 1980, however, if you have heart disease, you can still buy digitalis and diuretics at a price that has not even kept up with inflation. The difference today is that when any of those medications stop working you have a choice. You can get short of breath and die or you can place yourself in the hands of a cardiovascular surgeon and get a new valve or new coronary arteries put in at, of course, a great cost.

As another example, if you had renal failure in 1950, you didn't have to spend any money. You simply went on a specific diet and when your kidneys could no longer handle the problem you died. Today, there is an entirely different solution. You can have dialysis or a renal transplant which will prolong your life but at a far greater cost.



So what the President of the United States and so many congressmen point out about the cost of medicine, does not fit today's problem whatsoever. There is no need to be nostalgic about the price of medicine in 1950 because no one will buy that kind of medicine. We in medicine have now been faced with a very serious enigma. Medicine, by virtue of its spirit of scientific inquiry, has become a victim of its own success. It is villified and accused by the same public that demands new miracles, yet these miracles carry a larger and larger price tag.

It is a simple, inevitable fact that the more advanced health care becomes, the more expensive it becomes. No South Dakota physician in his right mind will revert to practicing medicine at the 1950 level. Patients will continue to demand an ever increasing success from us in diagnosing and treating their problems. As long as this trend continues, the bill for good health will continue to rise. The consumer will continue to want "Cadillac" care but will not obtain it at "Ford" prices. National Health Insurance is no answer and probably would negate many advances we have made in the past 30 years in diagnosis and treatment.

Sincerely yours,
Duane B. Reaney, M.D., President
South Dakota State Medical Association

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The South Dakota Medical School Endowment Association Needs You

G. E. Tracy, M.D.*

December has been designated "Endowment Month" by the Board of Directors of the South Dakota Medical School Endowment Association. The Board hopes that all physicians in South Dakota, graduates of the University of South Dakota School of Medicine and graduates of other schools, will become interested and involved in Endowment activities. This Association operates solely on contributions, and of course the greater the physician involvement and the more contributions received, the more effective the Association can be.

The Endowment Association was incorporated in November 1949 with the expressed purpose of providing support for the two year University of South Dakota School of Medicine. Since that time, the School of Medicine has expanded and is now a four year degree granting school. Along with this expansion, the Endowment Association's activities have increased and more requests for funds have been received. The Endowment Association provides loans to students at the University of South Dakota School of Medicine; such loans totalling approximately \$20,000 annually. In addition, \$2,000-\$4,000 is provided annually as matching monies to enable the school to obtain federal funds on a 9:1 matching

basis and this is not returnable to the Association. Also, the Endowment provides money for the Dean to help underwrite expenses incurred by him in conjunction with the school which are not reimbursible with state funding, and provides many other forms of assistance to the medical school.

This year the Endowment voted to assist the University of South Dakota School of Medicine in the establishment of an Alumni office in Vermillion and is providing partial underwriting of this office for a three year period. The Alumni office will work on alumni activities such as class reunions and will be soliciting donations from alumni, corporations and others to enhance programs and provide increased support for the University of South Dakota School of Medicine.

Both of these associations need and deserve physician support. Inflation has affected everyone, and it is anticipated that requests for student loans will increase as well as needs for the University of South Dakota School of Medicine. You will receive a reminder from the Endowment Association in the near future requesting your support. Please consider this request and send your tax deductible contribution to the South Dakota Medical School Endowment Association, 608 West Avenue, North, Sioux Falls, South Dakota. Help us to help your medical school and the students enrolled at the University of South Dakota School of Medicine.

President, Board of Directors, South Dakota Medical School Endowment Association.

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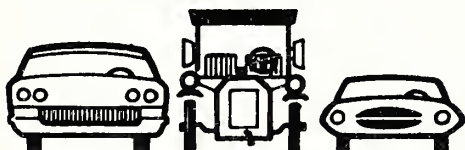
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Fifty-Two Year Old Man with Urethral Bleeding, Proteinuria, and Abnormal Serum Proteins

Milton C. Zadina, M.D.*
Discusser

John F. Barlow, M.D.**
Editor

CASE NO. 800388

This 52-year-old white Caucasian male was referred to Sioux Valley Hospital because of the recent onset of urethral bleeding of several weeks duration.

The patient had had three episodes of urethral bleeding over a two week period with no history of dysuria, frequency, or urethral trauma. There was no history of decreased urinary stream or difficulty voiding. He gave no history of previous kidney stones or present abdominal or flank pain. The bleeding seemed to occur at night.

The patient had had a previous history of peripheral thrombophlebitis and a pulmonary embolus for which he was anticoagulated several years prior to admission. Because of recent headaches, he had had a brain scan and electroencephalogram which were negative. The patient took acetaminophen and a compound containing barbiturate, codeine, caffeine, aspirin, and phenacetin for headaches (Fiorinal). There was no other significant history on review of systems.

PHYSICAL EXAMINATION: Height: 5' 8½"; pulse was 72 per minute and regular; respirations 20/minute and regular; blood pressure 120 systolic and 80 diastolic; temperature 98.6°F. The patient was a well-developed male appearing younger than his stated age. He was awake, alert, and fully oriented. Examination of the head and neck was un-

remarkable, except for a small white area above the left optic disc which could have represented a drusen body or cholesterol embolus. The thyroid was not palpable. The lungs were clear to auscultation and percussion. The heart was not enlarged and there were no murmurs or abnormal sounds. Pulses were full and symmetrical. The neurologic examination was within normal limits. There were no xanthomata present.

LABORATORY DATA: Routine urinalysis on admission: amber, hazy, specific gravity 1.033, pH 6.0, protein 4+, but negative for glucose, ketone bodies, bile, and hemoglobin; sediment 3-4 white cells/hpf, some oval fat bodies seen. In a subsequent urinalysis over 200 red cells were seen in the sediment. Hemoglobin 15.6 gm/dl with normal indices, total leukocytes 7,300/mm³ with 53% segmented neutrophils, 2% neutrophilic bands, 1% eosinophils, 5% basophils, and 39% lymphocytes. The red cells were unremarkable and the platelets were normal in number and morphology on smear. The platelet count was 225,000/mm³, and reticulocyte count 1.1%. Three LE preparations were negative. A fluorescent antinuclear antibody test (FANA) was negative; pH and electrolytes were within normal limits. Lactic dehydrogenase (LDH), alkaline phosphatase, aspartate aminotransferase (SGOT), total bilirubin, calcium, total protein, inorganic phosphorus, blood glucose, and uric acid were within normal limits. A creatinine was 1.5 mg/dl and a blood urea nitrogen 28 mg/dl. A creatinine clearance was 67 cc/mm. Triglycerides were 540 mg/dl (normal 30-190 mg/dl) and cholesterol 672 mg/dl (normal 100-290 mg/dl). Total protein was 6.3 gm/L with albumin of 3.1 gm/L, alpha I globulin 0.3 gm/L, alpha II globulin 1.1 gm/L, beta globulin 1.2 gm/L and gamma globulin 0.7 gm/L. There was a small monoclonal spike noted in the gamma globulin region. On serum and urine

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immunoelectrophoresis an IgG kappa monoclonal protein was present. The total urinary protein for 24 hours was 18.4 gm/L. Prothrombin time and partial thromboplastin time were within normal limits. A bone marrow biopsy was normally cellular with absent stainable iron and no increase in atypical plasma cells. A rectal biopsy was negative including special stains for amyloid. A computer axial tomogram of the brain showed some cerebral atrophy. A proctosigmoidoscopy was unremarkable. A cystoscopy and bilateral retrograde pyelograms were negative. An electrocardiogram showed normal sinus rhythm; a chest film was negative. Skull films showed slight carotid artery calcification. A diagnostic procedure was performed.

DR. ZADINA: In summary, the case under discussion today is that of a 52-year-old Caucasian male who had enjoyed generally good health until the recent onset of hematuria prompted his admission to the hospital for evaluation. His physical examination like his recent symptomatology was largely unremarkable. Subsequent laboratory workup, however, revealed extensive abnormalities in renal function and serum proteins which had previously been unsuspected.

Examination of the urine showed intermittent hematuria, a massive proteinuria of 18.4 gm/24 hours, and a moderate degree of renal failure reflected by a creatinine clearance of 67 cc/min. Electrophoresis showed intact IgG molecules in the urine. Examination of the blood revealed a corresponding abnormal IgG forming a monoclonal spike on serum protein immunoelectrophoresis. In addition the serum albumin and gamma globulin were noted to be in the lower ranges of normal. Examination of the bone marrow showed no increase in atypical plasmacytes.

In short, the disease entity present in this individual must be one which contains both production of a monoclonal IgG and renal failure within its spectrum of manifestations. There are only two such diseases which can present in this fashion, these being multiple myeloma and amyloidosis.

Prior to discussing these disease entities I would like to briefly review immunoglobulin structure and terminology. Immunoglobulins are composed of two light and two heavy protein chains bound together by disulfide bonds. Each chain possesses a variable and a constant end. The variable end determines the antigenic specificity of the immunoglobulin. The constant end of the heavy chain determines which of the five classes of immunoglobulins (IgG, IgA, IgM, IgD, IgE) to which the molecule belongs. The constant end of the light chain determines whether it is a kappa or a lambda light chain. Plasma cells are the cells of origin for these humoral antibodies. When an abnormal clone of plasma cells produces a homogenous protein which appears as a spike on serum or urine electrophoresis, that abnormal pro-

tein is referred to as an M-component (M standing for monoclonal). M-components can consist of either whole immunoglobulins of any class or of either class of light chains only. Light chains are generally produced in slight excess of heavy chains and these light chains may then appear in the urine after filtering through the glomerular membrane. When these small chains appear in the urine, they are referred to as Bence Jones proteins.

One disease in which M-components play a major role is multiple myeloma. Multiple myeloma is a neoplasm of plasma cells. It generally occurs in elderly people with a peak in incidence between the ages of 60 and 70. It is slightly more common in males than in females and has a general incidence of 1-2/100,000. The natural history of the disease is one of steady deterioration. The patient first goes through an asymptomatic period where the only abnormalities might be an elevated erythrocyte sedimentation rate (ESR), the presence of an M-component in the serum, and Bence Jones proteinuria. Eventually, however, nonspecific symptoms such as weakness, weight loss, and an increased susceptibility to infection become apparent. Ultimately, the patients develop skeletal or renal symptomatology.

The skeletal lesions are the hallmark of the disease and generally present with localized swelling, tenderness, or pathological fracture. On x-ray the bony lesions appear as lytic or punched out areas and evidence of periosteal reaction or new bone formation is rare. Early in the course of the disease a diffuse osteoporosis may be the only finding.

Examination of the blood generally reveals a normochromic, normocytic anemia with hemoglobin values between 7 and 10 gm/dl. Reticulocyte count is generally low. Rouleaux formation is common secondary to the increased globulin in the serum and is reported in approximately 75% of cases. An increased ESR is also common. Up to 50% may have a mild decrease in neutrophils or increase in lymphocytes but total white blood cell counts tend to be within normal limits. Occasionally, an immature plasma cell may be spotted on peripheral smear. Platelets are usually normal in number.

Serum lipids are usually low or normal in multiple myeloma but occasionally cases of myeloma are reported with very high serum lipids, often with accompanying xanthelasmas.³ Our patient is noted to have high triglycerides and cholesterol in the absence of frank nephrotic syndrome and this is not inconsistent with multiple myeloma.

Protein abnormalities are very common in the serum and urine and over 99% of patients with myeloma secrete some type of homogenous immunoprotein. The protein secreted can be of any immuno-

globulin class or it may consist of light chains only. Approximately 50% of patients with multiple myeloma will secrete IgG, 25% will secrete IgA, 24% light chains only, 1% IgD, and IgM and IgE are reported only rarely. If light chains only are secreted, they will often not give a serum spike because of rapid filtration through the glomeruli but will appear in the urine as Bence Jones proteins.

The clinical picture can vary somewhat with the particular immunoglobulin excreted. IgG disease, as is present in this patient, has classically been associated with generalized decrease in serum immunoglobulins, a markedly increased susceptibility to infections, large quantities of M-component in the serum, a relatively low serum calcium as compared to other types of myeloma and also a slightly decreased incidence of amyloidosis.

The renal disease associated with multiple myeloma is basically a tubular disorder. There are a number of factors present which are felt to contribute to this problem. First of all, filtration of Bence Jones proteins by the glomeruli leads to a massive protein resorption load in the renal tubules which can lead to tubular degeneration and decreased tubular function. Casts play a major role in the disease in that they plug the tubular lumen and can lead to dilatation and destruction of the nephron. Tubular dysfunction is occasionally reflected by low specific gravity and polyuria. Hypercalcemia and hyperuricemia if present can contribute to the renal problem. Glomerular filtration rate can be decreased but generally this is in conjunction with a decreased renal plasma flow. Glomerular involvement is not a major factor in the disease, and the proteins spilled in the urine are, therefore, mainly Bence Jones proteins which are small enough to pass through the glomerular filter without any antecedent damage being necessary to allow their passage. It is felt that the presence of large proteins in the urine cannot be attributed to uncomplicated myelomatous involvement of the kidney.⁶ In fact, it has been argued that even the passage of albumin is so uncharacteristic of a strictly myelomatous kidney that a complicating renal factor must be considered.

Various criteria for the diagnosis of multiple myeloma have been proposed. These criteria include cytologic and laboratory data. The cytologic criteria are either plasma or myeloma cells over 10% when 1,000 cells are counted in the marrow or biopsy, proven plasmacytoma in bone, or soft tissue. The laboratory criteria are myeloma protein in plasma and/or urine, x-ray evidence of osteolytic lesions, or myeloma cells present on peripheral smears. To make the diagnosis of multiple myeloma you need both cytological criteria or one cytological and one

laboratory criteria.¹¹ It is important to realize, however, that one bone marrow is not necessarily enough to rule out myeloma since the marrow involvement is often spotty and a low plasma cell count may simply reflect a relatively uninvolved area.

On the basis of the criteria outlined above, it is unlikely that this patient has multiple myeloma. It is stated that there is "no increase in atypical plasma cells" but no percentage of bone marrow involvement is given. In addition, the patient is missing some of the other key manifestations of multiple myeloma. There is no indication of the patient feeling particularly ill in the history and skeletal pain is not mentioned. On laboratory analysis, rouleaux formation is not noted on the blood smear and anemia is absent. In addition, the urine is atypical for multiple myeloma in two important aspects. First of all, no casts are seen in the urinary sediment and casts appear to play a major role in the pathogenesis of myeloma kidney. Secondly, there is massive proteinuria containing IgG molecules as part of the protein fraction. IgG is a large molecule and its presence in the urine strongly suggests severe glomerular damage which is not a feature of uncomplicated myelomatous disease. Therefore, although the presence of the M-component in urine and serum is highly suspicious for possible myeloma, one must also look for some other concomitant process to explain the urinary findings. This leads us to the consideration of amyloidosis.

Amyloidosis is a disease process associated with the finding of homogenous eosinophilic material in various organs of the body. The eosinophilic material if stained with Congo Red and viewed under polarized light gives off a characteristic green birefringence which is felt to be a finding specific for the disease. When viewed under the electron microscope, this amyloid material is found to be made up of crossing non-branching fibrils which in turn are made up of two filaments and arranged in an antiparallel beta pleated sheet configuration.

Historically, amyloidosis has been a difficult and confusing disease to work with. This has stemmed from difficulty in analyzing the amyloid material chemically. This, until recently, has stymied efforts to elucidate its nature beyond the fact that it is a protein. Lacking a clear cut pathogenesis, classification of the disease has largely been based on clinical manifestations.

Traditionally, amyloidosis has been separated into two clinical categories namely primary and secondary amyloidosis. Primary amyloidosis was felt to occur in the absence of any underlying disease processes and the disease was felt to involve mainly organs such as the heart, tongue, skeletal muscles,

and blood vessels. Secondary amyloidosis was described as being found in patients with a chronic underlying infection (osteomyelitis, bronchiectasis, tuberculosis, leprosy) or inflammatory process (rheumatoid arthritis, ulcerative colitis). The secondary type of amyloidosis was thought to involve mainly liver, spleen, kidney, and adrenals in its organ distribution.

The concepts of primary and secondary amyloidosis have survived to the present day but have been modified considerably. First, it has been found that the organ distributions classically described do not hold up. Renal involvement, for instance, has been found to be just as common in primary disease as in secondary amyloidosis. Secondly, several other types of amyloidosis which do not readily fit under either the primary or secondary categories have been discovered.

The most important of the newer types of amyloidosis described is that which is associated with plasma cell dyscrasias. As methods for the detection of M-components in urine and serum become more and more refined, an increasing number of amyloid cases are found to have an associated plasma cell dyscrasia. This type of amyloidosis had variously been listed as a separate entity or as a subgroup of primary amyloidosis since its tissue distribution seemed to be most consistent with primary disease. The most recent work, however, shows that a large percentage of all types of amyloidosis have M-components present. A recent study of 100 patients with all types of amyloidosis showed 88 of 100 patients studied to have an M-component in urine or serum. The M-component was found in 50 of 50 cases of primary amyloidosis, 9 of 17 cases of secondary amyloidosis and 26 of 30 cases of mixed amyloidosis.⁷

Various types of familial amyloidosis have also been described which have a wide variety of clinical features and do not readily fall into either primary or secondary categories. Localized amyloidosis has been increasingly recognized and seems to be a separate clinical subgroup. It is characterized by the finding of a localized collection of amyloid without apparent systemic involvement. Another recently recognized form of amyloidosis is that associated with aging. In people over age 70, amyloid can be found in the heart of 31%, in the aortas of 50%, in the pancreases of 30%, and in the brains of 67% of patients.

Recently, investigators have succeeded in solubilizing amyloid fibril proteins in the laboratory and have been able to perform amino acid sequencing on the proteins obtained in an effort to elucidate their origin. They have found that there are basically two

types of amyloid with two different origins.

In the majority of patients with primary amyloidosis, the predominant amyloid protein has been found to be a fragment of a monoclonal Bence Jones protein. The fragment of the light chain involved is the variable end and only certain Bence Jones variable ends have the physical properties necessary for the eventual formation of amyloid beta pleated sheets. The variable end proteins that possess these properties are referred to as amyloidogenic. The pathogenesis for amyloidosis postulated from this data is that abnormal plasma cells secrete large amounts of abnormal monoclonal immunoglobulin proteins which are carried in the serum to the cells of the reticuloendothelial system which take up the abnormal proteins and destroy them. In the process of metabolism the light chains are divided into their constant and variable portions and those variable ends, which have the physical properties needed for the formation of beta pleated sheets, will then go on to form amyloid fibrils which are deposited into the surrounding tissues.

The second type of amyloid protein has been elucidated in a fair number of other patients and has been dubbed AUO (amyloid of unknown origin). This protein has a relatively constant structure from patient to patient and occurs mainly in those with secondary and senile forms of the disease. Since the origin of AUO is still being sought, the pathogenesis of this type of amyloid is still unknown. However, an antisera to AUO has been developed which cross reacts with an as yet unknown serum factor in 7% of normal people and approximately 50% of those with chronic underlying disease. It has thus been postulated that AUO is probably derived from a normal serum protein; which, for some reason, is elevated in those disorders associated with secondary amyloidosis.

Thus, it would seem that a new classification of amyloidosis might involve one category consisting of amyloid derived from immunoglobulin light chains and one category consisting of deposits derived from AUO. Unfortunately, even this classification is lacking in specificity since immunoglobulin derived protein has been found in amyloid composed primarily of AUO and AUO has been found in amyloid associated with M-components and composed chiefly of immunoglobulin protein.

Clinically, amyloidosis can have a myriad of associated signs and symptoms depending on which organ systems are involved. Common sites of involvement are kidney, heart, gastrointestinal tract, liver, respiratory tract, nervous system, eye, skin, spleen, thyroid, adrenal, and pituitary.

There is no laboratory test which is specific or

unique for the disease. Generally there is an increased ESR and fibrinogen is elevated. Serum complement may be decreased and a deficiency of coagulation factor X has been reported. Examination of serum protein may show hypogammaglobulinemia and 88% will have an M-component. Bence Jones protein in the urine and plasmacytosis in the marrow are common findings. In addition, recently articles have appeared which state that amyloid fibrils can be identified in the urinary sediment of patients with renal amyloidosis when viewed under the electron microscope.⁴

Of all the clinical and laboratory findings those related to amyloidosis of the kidney are the most common and most serious manifestations of the disease. The kidney is the most common presenting organ with disease and renal failure is the most common cause of death. The basic pathological process in the kidney is the gradual replacement of the glomeruli with amyloid deposits. Note that this is in direct contrast to myelomatous disease of the kidney which is mainly a tubular degenerative process. Because the glomeruli are the site of involvement, the findings are those you would expect to find in a glomerular disease, namely proteinuria and persistent hematuria. The proteinuria can be present in massive amounts and up to 60% of patients will eventually develop the nephrotic syndrome. Early in the process, however, hypertension is rare and massive proteinuria can occur in the absence of the nephrotic syndrome. Since the damage to the glomeruli can be quite extensive, it is not unusual to lose entire immunoglobulins through the glomerular filter into the urine. In addition, albuminuria is very common.

I feel that the urinary findings in this patient are more consistent with amyloidosis than with multiple myeloma. The presence of gamma globulin in the urine is suggestive of severe glomerular damage which is rare in a purely myelomatous kidney and the absence of casts in the sediment also mitigates against a myelomatous process.

Unfortunately, the diagnosis of amyloidosis is not always an easy one to make premortem. One diagnostic test used in the past was the Congo Red extraction test. In this study a measured amount of Congo Red was injected into the bloodstream and allowed to equilibrate for a brief period of time. A serum sample was then drawn and the percentage of dye extracted from the serum by the body tissues was calculated. Because Congo Red has such a great affinity for amyloid, it was felt that a high extraction

percentage would be a good indicator for the presence of amyloid in the subject. Unfortunately, the test gave an unacceptable number of false positive and especially false negative results and also was associated with a fair percentage of anaphylactoid reactions after injection of the dye. For all the above reasons, the test has been largely abandoned. The most widely accepted method for diagnosis at this time is via biopsy and appropriate staining techniques looking for green birefringence under polarized light after staining with Congo Red. A wide variety of tissues have been biopsied attempting to make the diagnosis. Rectal biopsies are reported to be positive in 75% of patients with amyloidosis and gingival biopsies are positive in 60%. Biopsy of the skin is sometimes positive even in the absence of visible lesions. Biopsy of kidney or liver is also positive in a large percentage especially if clinical signs or laboratory data support involvement of those organs. Closed biopsy of liver or kidney is less preferred than biopsy of more readily accessible areas because of the tendency for markedly amyloid laden organs to bleed. Indeed, some feel that biopsy of a grossly enlarged liver in suspected amyloidosis is contraindicated due to the danger of hemorrhage.

I strongly suspect the diagnostic procedure performed in this case was renal biopsy as that is the primary organ of concern in this case. I would expect it to show amyloid accumulation in the glomeruli.

Dr. Zadina's Diagnoses Amyloidosis Associated With A Plasma Cell Dyscrasia

DR. BARLOW: Dr. Zadina is right in that a renal biopsy was performed. The tubules did not show tubular casts and there were only rare areas of interstitial inflammation. The major seat of the disease was in the glomeruli where there was focal eosinophilic thickening of the capillary walls (Figure 1). Special stains including Congo Red for amyloid were not diagnostic. However, because of an appearance suggestive of amyloid, an electron micrograph was performed on the tissue from paraffin block. On this slide (Figure 2) the typical fibrillar appearance of amyloid can be seen.

*DR. RICHARD A. JAQUA: Since the material was obtained from the paraffin block the cellular detail of the remainder of the glomerulus is not very good but the diagnosis can still be made. I would like to point out that the electron micrographic appearance of amyloid is the same with the electron microscope even though it can have several origins, as Dr. Zadina has suggested. He also mentioned the fact that electron micrographic studies of urine have

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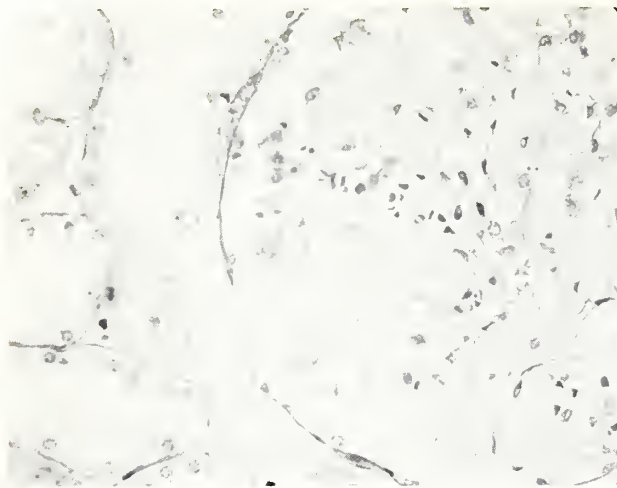


Figure 1
Thin section showing nodules of hyalin material in glomerular tuft.

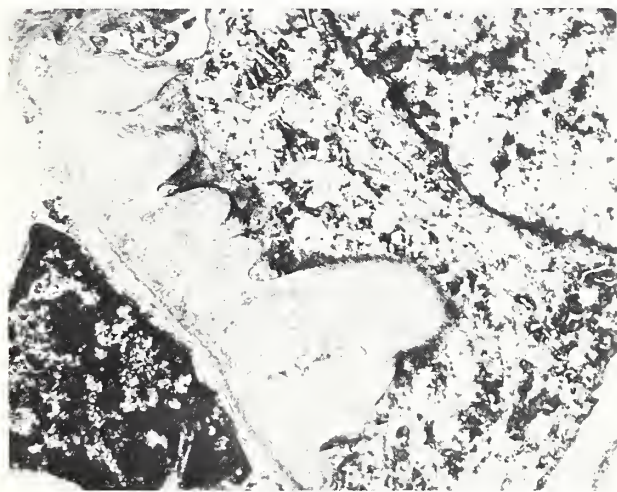


Figure 2
Light staining structure in center shows fibrillar pattern of amyloid by electron microscopy.

been attempted to make a diagnosis of amyloidosis. Unfortunately, there are many substances in the urine which can give a structural appearance similar to amyloid. This makes urine sediment examination very difficult. I am afraid that the diagnosis of amyloidosis by the examination of urinary sediment with the electron microscope is not going to prove to be helpful.

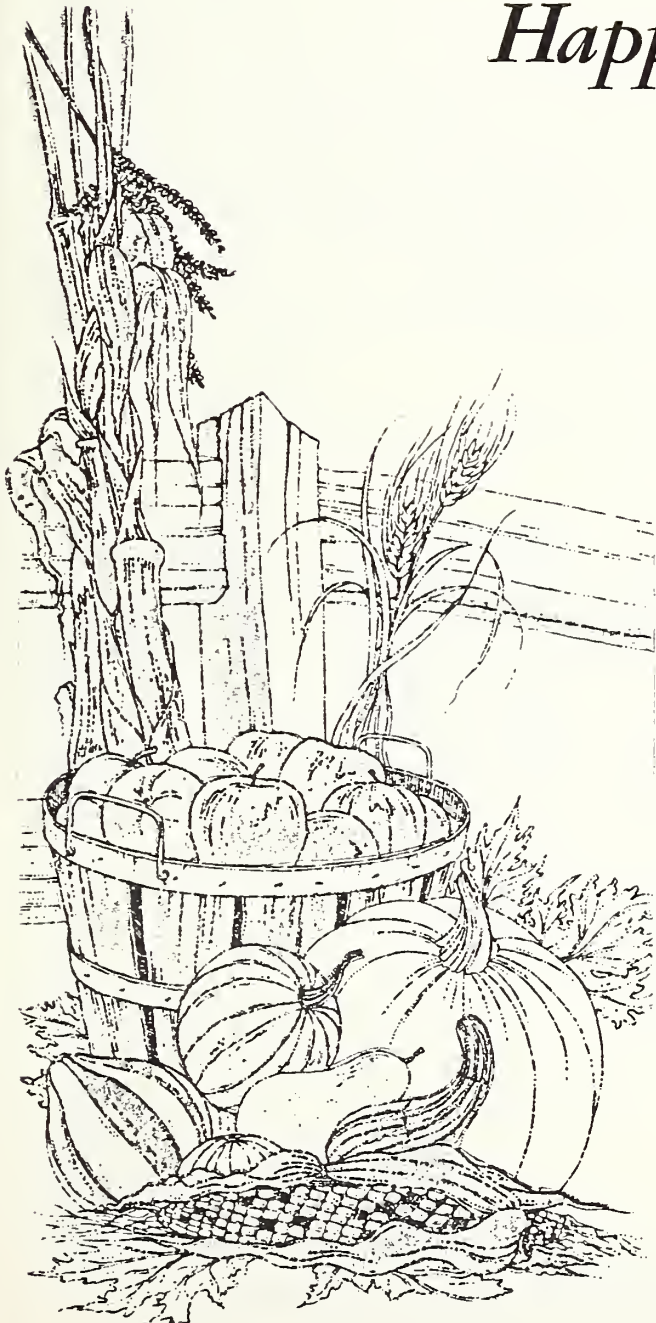
FINAL ANATOMIC DIAGNOSIS PRIMARY AMYLOIDOSIS INVOLVING THE KIDNEYS

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New Names For Old Diseases

Recently there has been an increasing tendency to replace the old terminology for precursor lesions of invasive carcinoma of the cervix (from varying degrees of dysplasia up to carcinoma in situ) with a new terminology called Cervical Intraepithelial Neoplasia (CIN) with grades I, II, and III. The reasons for this are as follows:

1. More recent thought has suggested that the various degrees of dysplasia and carcinoma in situ represent a continuum, which may culminate in invasive carcinoma. The continuum theory has been supported by (A) The trend of increasing age with the more severe lesions which still tend to precede the age group in which invasive carcinoma is seen. (B) The reversibility of the atypical changes in the cervical epithelium as compared to its tendency to develop increasing atypia or become invasive has been shown to be related to the degree of the abnormality in the epithelium when first noted. In other words, the more atypical the lesion appears, including carcinoma in situ, the less likely the lesion is to regress, and the more likely the lesion is to be-

come invasive. Therefore, to use different terms such as dysplasia and carcinoma in situ, which do not connote any sort of continuum can be misleading.

2. The use of dysplasia (implying a nonmalignant lesion), and carcinoma in situ (implying a malignant lesion) for entities which have great histological similarity is probably not warranted.

3. It has been noted in many studies that the distinction between severe dysplasia and carcinoma in situ is difficult and not a reproducible diagnosis amongst a number of pathologists. This leads to confusion by the clinician when one pathologist calls the lesion carcinoma in situ and a second pathologist or consultant calls the lesion severe dysplasia. Although the two are extremely similar histologically, and both connote a proclivity for progression to invasive carcinoma, the implication is clear from the term carcinoma in situ, but less so from the word dysplasia. When terminology such as CIN is used, a continuum is implied and distinctions which are at best difficult to make are not overemphasized. Below is a comparison between the new and old terminology:

New Terminology	Old Terminology
Cervical Intraepithelial Neoplasia (CIN)	Dysplasia — carcinoma in situ
CIN, Grade I	Very mild and mild dysplasia
CIN, Grade II	Moderate dysplasia
CIN, Grade III	Severe dysplasia and carcinoma in situ
Microinvasive carcinoma	Microinvasive carcinoma

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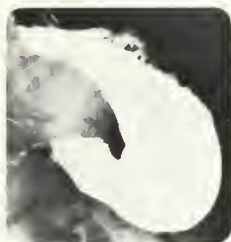
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"The correlation of spasm relief and drug given was excellent."

*This drug has been classified "probably" effective in treating functional bowel/irritable bowel syndrome.

†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:

King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

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Brief Summary

INDICATIONS

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the following indications as "probably" effective.

For the treatment of functional bowel/irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

THESE FUNCTIONAL DISORDERS ARE OFTEN RELIEVED BY VARYING COMBINATIONS OF SEDATIVE, REASSURANCE, PHYSICIAN INTEREST, AMELIORATION OF ENVIRONMENTAL FACTORS.

For use in the treatment of infant colic (syrup).

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloro-duodenal stenosis); paralytic ileus, intestinal atony of the elderly or debilitated patient, unstable cardiovascular status in acute hemorrhage, severe ulcerative colitis; toxic megacolon complicating ulcerative colitis; myasthenia gravis. **WARNINGS:** In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. **PRECAUTIONS:** Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy. Use with caution in patients with: Autonomic neuropathy. Hepatic or renal disease. Ulcerative colitis. Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon. Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension. Hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.

Do not rely on the use of the drug in the presence of complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. **ADVERSE REACTIONS:** Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia; urinary hesitancy and retention; blurred vision and tachycardia; palpitations; mydriasis; cycloplegia; increased ocular tension; loss of taste; headache; nervousness; drowsiness; weakness; dizziness; insomnia; nausea; vomiting; impotence; suppression of lactation; constipation; bloated feeling; severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating. With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. **DOSAGE AND ADMINISTRATION:** Dosage must be adjusted to individual patient's needs.

Usual Dosage: Bentyl 10 mg. capsule and syrup: Adults: 1 or 2 capsules or teaspoonfuls syrup three or four times daily. Children: 1 capsule or teaspoonful syrup three or four times daily. Infants: ½ teaspoonful syrup three or four times daily. (May be diluted with equal volume of water.) Bentyl 20 mg. Adults: 1 tablet three or four times daily. Bentyl Injection: Adults: 2 ml. (20 mg.) every four to six hours intramuscularly only. NOT FOR INTRAVENOUS USE. **MANAGEMENT OF OVERDOSE:** The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal. Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested. If indicated, parenteral cholinergic agents such as Urecholine[®] (bethanechol chloride USP) should be used.

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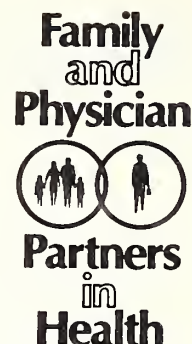
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1979 Assembly in Atlanta:

SDAFP Delegates R. W. Friess, M.D. and L. H. Amundson, M.D. represented our chapter at the annual national meeting. Final registration for the Academy's 31st Annual Scientific Assembly totaled 8,156. This number included 3,523 physicians, 3,183 guests, 1,224 technical exhibitors and 226 scientific exhibitors.

SD

Future Meetings

November

AAMI/FDA Conference on Medical Device Regulation, Hyatt-Regency Hotel, Washington, D.C., Nov. 27-28. Fee: \$180. Contact: AAMI, Box 460, Springfield, VA 22150. Phone: (703) 525-4890.

Radiation Therapy Seminar, U. of Iowa Coll. of Med., Iowa City, IA, Nov. 30. AMA Category 1 credits. Contact: Richard Caplan, M.D., Asso. Dean for Cont. Med. Ed., U. of Iowa Coll. of Med., Iowa City, IA 52242.

Otolaryngology Clinical Conference, U. of Iowa Coll. of Med., Iowa City, IA, Nov. 30. AMA Category 1 credits. Contact: Richard Caplan, M.D., Asso. Dean for Cont. Med. Ed., U. of Iowa Coll. of Med., Iowa City, IA 52242.

December

Cardiology Today, U. of Iowa Coll. of Med., Iowa City, IA, Dec. 3-6. AMA Category 1 credits. Contact: Richard Caplan, M.D., Asso. Dean for Cont. Med. Ed., U. of Iowa Coll. of Med., Iowa City, IA 52242.

Emergency Procedures for Physicians, U. of Iowa Coll. of Med., Iowa City, IA, Dec. 5. AMA Category 1 credits. Contact: Richard Caplan, M.D., Asso. Dean for Cont. Med. Ed., U. of Iowa Coll. of Med., Iowa City, IA 52242.

Ophthalmology Clinical Conference, U. of Iowa Coll. of Med., Iowa City, IA, Dec. 5. AMA Category 1 credits. Contact: Richard Caplan, M.D., Asso. Dean for Cont. Med. Ed., U. of Iowa Coll. of Med., Iowa City, IA 52242.

The Fourth Southeastern Conference on Alcohol and Drug Abuse, Atlanta, GA, Dec. 5-9. 27 hrs. Category 1 credits. Contact: Conway Hunter, M.D., Peachford Hosp., 215 Peachford Road, NE, Atlanta, GA 30338. Phone: (404) 455-3200.

Care of Burned Patient, Towsley Ctr. for CME, U. of Mich., Ann Arbor, MI, Dec. 7. Contact: Off. of CME, U. of Mich. Med. School, Towsley Ctr. for CME, Ann Arbor, MI 48109. Phone: (313) 764-2287.

Clinical Nutrition for the Practicing Physician, Sheraton-Ritz Hotel, Minneapolis, MN, Dec. 7-8. 10 hrs. AMA Category 1 credits. Contact: CME Off., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Continuing Pathology, Towsley Ctr. for CME, U. of Mich., Ann Arbor, MI, Dec. 11. Contact: Off. of CME, U. of Mich. Med. School, Towsley Ctr. for CME, Ann Arbor, MI 48109. Phone: (313) 764-2287.

Workshop on Coronary Heart Disease, Spring Hill Ctr., Wayzata, MN, Dec. 12-14. 17 hrs. AMA Category 1 credits. Contact: CME Off., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

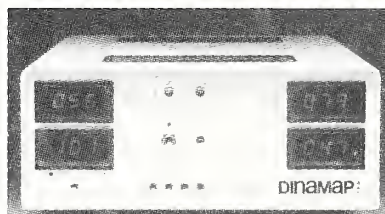
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**A Study of Endometrial Carcinoma and
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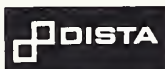
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Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed, drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. **Adults:** Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. **Geriatric or debilitated patients:** 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) **Children:** 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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SCIENTIFIC ARTICLES

- 5** A Study of Endometrial Carcinoma and
Potential Precursor Lesions (Part I)
Brooks Ranney, M.D.
- 21** Clinicopathological Conference
Fifteen Year Old Male and Six Month Old
Male with Marked Hypoproteinemia and Edema
Kurt E. Johnson, M.D.
John D. Barker, M.D.
John F. Barlow, M.D.

FEATURES

- 15** President's Page
- 17** South Dakota AFP Chapter News
- 18** Laboratory Aids
South Dakota Pathology Page
- 29** Council Meeting Highlights
- 31** Index to Volume XXXII
- 36** Future Meetings

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A Study of Endometrial Carcinoma and Potential Precursor Lesions (Part I)*

Brooks Ranney, M.D.**

ABSTRACT

A 30-year study of patients who had endometrial carcinoma or precursor lesions disclosed that these conditions were found in 3.37% of a patient population of 38,300 mature women, or in 19.86% of the 6,491 women who needed gynecologic operative procedures.

Among 515 patients with endometrial polyps, 222 exhibited spotting bleeding, and 9.91% of these also had endometrial carcinoma.

Among 615 patients with cystic endometrial hyperplasia, only 1.53% subsequently had endometrial carcinoma. However, more than two-fifths of the 84 patients who had endometrial carcinoma also showed microscopic cystic hyperplasia of the endometrium.

Among the 75 patients with adenomatous endo-

metrial hyperplasia, 20% had present or subsequent endometrial carcinoma.

However, among the 84 patients with endometrial cancer, the cancer had developed in otherwise atrophic endometria in 19 older patients.

History, symptoms, potential etiologic factors, diagnostic methods, findings, staging, methods of treatment, prognosis, and survival have been studied and are discussed. Probably the most important factors affecting survival are (1) early, meticulous diagnosis, (2) prompt operative extirpation (avoiding spillage or extrusion of cancer cells during removal), and (3) the host-resistance of each individual patient. Depth of myometrial invasion in the microscopic sections constitutes the best index of prognosis—deeper invasion, poorer prognosis.

INTRODUCTION

Proliferative and malignant lesions of the endometrium have produced special problems for the gynecologist. The results of early diagnosis and adequate therapy are generally very good.^{9,10,15,18,19} Yet

there is a small, elusive group of patients who rapidly succumb to endometrial cancer, probably because of poor host-resistance.^{10,15,20,32,36,51} Likewise, there is another tiny group of patients whose distant metastases may first become evident some years after initial therapy. Possibly, because of good host-resistance, these few patients have lived for years in symbiosis with cancer.^{15,20,32,36,51}

This study was designed to seek answers to the following questions:

1. What endometrial lesions may precede, or be associated with endometrial carcinoma? How

* Presented at the South Dakota Section of the American College of Obstetricians and Gynecologists in Yankton, South Dakota on May 10, 1979. This is Part I of a two part series.

** Department of Obstetrics and Gynecology, University of South Dakota School of Medicine, The Yankton Clinic, 400 Park, Yankton, South Dakota 57078.

often? What are the time-sequential relationships between potential precursor lesions and endometrial cancer?

2. Which potentially etiologic factors are most frequently present in the medical history of patients who have endometrial carcinoma? Which symptoms occur most frequently?

3. Which methods of diagnosis and treatment are best adapted to which stages of endometrial cancer? What are the results of treatment? Which "precursor" lesions require immediate definitive operations? How many patients with "precursor" lesions need later definitive operations?

4. How may one estimate prognosis for patients with endometrial cancer? What is the long-term survival?

MATERIALS AND METHODS

During the years 1948 through 1978, approximately 206,000 gynecologic examinations have been performed by the author upon patients in the office, from among a population of about 38,300 female patients of reproductive age, or older. Of these, 3,157 patients needed minor gynecologic operative procedures, and 3,334 patients needed major gynecologic operative procedures—total, 6,491.

Gross or microscopic abnormalities of the endometrium were observed during 1,289 (19.86%) of these operative procedures, or in 3.37% of the patient population of 38,300 women.^{12,39,45} These abnormalities may be classified generally, as noted in Table I. A small number of patients had two or more associated abnormalities. Most of these abnormalities had caused symptoms, chiefly "spotting" bleeding.

Individual office records, operative reports, pathology reports, and punch cards from these patients have been studied.

Microscopic sections from patients with adenomatous hyperplasia, and patients with endometrial cancer have been reviewed. Cancer patients have been staged according to FIGO categories (Addendum A), both gross and microscopic. Additionally, endometrial cancers were grouped according to depth of penetration of the myometrium^{6,9,14} as noted in Table II.

Follow-up information from periodic examinations of most of these patients was available for intervals ranging from one to 30 years after operation. A few patients were traced through the tumor registry. Information is complete for all patients who had adenomatous hyperplasia or endometrial cancer.

Table I
Classification of
Endometrial Abnormalities

Classification	Number	Percent
Endometrial polyps	515	39.95
Cystic hyperplasia	615	47.71
Adenomatous hyperplasia	75	5.82
Cancer of endometrium	84	6.52
	1289	

Addendum — A:

FIGO Stages of Endometrial Carcinoma

Stage 0—Microfocal malignant lesions confined to endometrium

Stage I—Carcinoma confined to corpus

Stage I,a—Uterine lumen, 8 cm. or less

Stage I,b—Uterine lumen, more than 8 cm.

G₁—Differentiated, glandular

G₂—Moderately differentiated, some solid

G₃—Mostly solid; undifferentiated

Stage II—Carcinoma involving both corpus and cervix

Stage III—Carcinoma has extended outside uterus, but not outside pelvis

Stage IV—Carcinoma has extended outside pelvis, or has involved bladder or rectum

Table II

Depth of Penetration of Endometrial Cancer

Group 1—cancer limited to the endometrium

Group 2—cancer infiltrates only the medial third of myometrium

Group 3—cancer infiltrates the middle third of the myometrium

Group 4—cancer spreads to the lateral third of the myometrium, to the serosa, and beyond

RESULTS AND DISCUSSION

1. Endometrial Polyps

Among the 6,491 patients who needed gynecologic operations, 515 (7.93%) were discovered to have endometrial polyps.^{3,16,52}

Presently, asymptomatic polyps were found in hysterectomy specimens from 293 patients (56.9%). However, among these, 121 (41.3%) had had curettage previously to search for the cause of earlier spotting. The other 172 patients (58.7%) gave no history of either prior or present symptoms.

Spotting bleeding from the uterus necessitated curettage and excision of endometrial polyps in 222 patients—43.1% of all 515 patients who were found to have polyps. Most of these polyps and endometria

were benign, but 16 patients had endometrial cancer either in the polyps or in other endometrial surfaces—3.1% of all 515 patients who had polyps, or 7.2% of the 222 patients who had spotting bleeding with endometrial polyps.

After curettage and excision of endometrial polyps, and hysterectomies, 206 patients retained uteri. Within one to seven years (average 4.3 years), 22 of these patients had recurring spotting and needed curettage. Six of these patients were found to have endometrial cancer (2.91% of the 206 patients who retained uteri after excision of endometrial polyps, or 27.3% of those patients who had recurring spotting).

Another 62 patients, from the 206 who retained uteri after curettage and excision of endometrial polyps (30%), needed hysterectomy during later years for treatment of other pelvic abnormalities, such as myomas, adenomyosis, endometrial hyperplasia, prolapse, neoplasia of the cervix, neoplasia of the ovaries, endometriosis.

In summary, no asymptomatic endometrial polyp was discovered to be associated with endometrial cancer. However, among the 222 patients who had spotting bleeding and endometrial polyps, 22 (9.91%) also had endometrial cancer in or adjacent to the polyps, or developed endometrial cancer within one to seven years. Another 62 patients needed later hysterectomy and other indicated gynecologic operative procedures for the reasons noted above. Only 138 (62.16%) did not require any gynecologic operation within the next one to 30 years after curettage and excision of endometrial polyps.

2. Cystic Endometrial Hyperplasia

Among 6,491 patients who needed gynecologic operations, 615 (9.47%) were found to have cystic hyperplasia of the endometrium.^{16,52}

Presently, asymptomatic cystic hyperplasia was observed in microscopic sections of hysterectomy specimens from 223 patients (36.26%). However, among these, 188 patients (52.91%) had had curettage previously to search for the cause of earlier spotting. The other 105 patients (47.09%) gave no history of present or prior symptoms.

Spotting bleeding from the uterus necessitated curettage for diagnosis in 392 patients—63.74% of all 615 patients who were found to have cystic hyperplasia of the endometrium. No cancers were found.

However, among these 392 patients who retained uteri after curettage for cystic hyperplasia, 59 patients (15.1%) later had spotting and needed another diagnostic curettage within one to ten years (average 6.5 years). This second specimen also dis-

closed endometrial cancer in six patients (1.53% of the 392 patients who retained uteri after diagnostic curettage for cystic hyperplasia, or 10.2% of those 59 patients who had recurring spotting).

Another 171 of these 392 patients who retained uteri after diagnostic curettage for cystic hyperplasia of the endometrium (43.63%) later needed hysterectomy and other indicated gynecologic operations for treatment of other pelvic abnormalities, such as myomas, adenomyosis, prolapse, neoplasia of the cervix, neoplasia of the ovaries, endometriosis.

In summary, no patient whose major microscopic diagnosis was cystic hyperplasia of the endometrium was found to have endometrial cancer at initial curettage, but six patients subsequently developed endometrial cancer (1.53% of those who retained uteri, or 10.2% of those who had second spotting). Likewise, 171 other patients needed hysterectomy for other reasons listed above. Only 215 patients (54.85%) did not require any gynecologic operation within the next one to 30 years after initial diagnostic curettage for cystic hyperplasia of the endometrium.

3. Adenomatous Hyperplasia of the Endometrium

Among 6,491 patients who needed gynecologic operations, 75 patients (1.16%) were found to have adenomatous hyperplasia of the endometrium.^{13,16,18,19,52}

a. Follow-up

All of these patients have been evaluated since then by periodic pelvic examinations, from one to 30 years, until the present, or until death. Ten patients have died an average of 10.1 years after adenomatous hyperplasia of the endometrium was found following curettage. Causes of death are listed in Table III. Most notable are two deaths, due to cancer of the breast and cancer of the ovary, re-

Table III
Interval Since Curettage, and Cause of Death
(Patients who had
Adenomatous Hyperplasia Endometrium)
(10 Patients)

Number of years after curettage	Cause of Death	Age at Death
5	Stroke	50
15	Stroke	75
6	Cardiac decompensation	69
15	Cardiac decompensation	90
20	Renal failure	89
10	Pulmonary embolus	55
2	Carcinoma of stomach	64
4	Carcinoma of breast	59
6	Carcinoma of ovary	57
18	Carcinoma of colon	65

spectively, four and six years after the curettage which had been performed for adenomatous endometrial hyperplasia. The remaining 65 patients have had full follow-up to the present, for an average of 11.53 years.

b. Age

When adenomatous hyperplasia of the endometrium was discovered, the youngest patient was 35, and the oldest was 81 years old (Table IV). Most patients (73.3%) were between 40 and 60.¹⁸

Table IV	
Age When Adenomatous Hyperplasia of Endometrium Was First Discovered	
Age Range	No. of Patients
35-39	9
40-49	32
50-59	23
60-69	8
70-79	2
80 +	1

c. History

Among these 75 patients, 22 (29.3%) were nullipara, and four others had had only one pregnancy—a much lower-than-average fertility. Only 11 patients (14.6%) had any history of prior usage of oral or injectable estrogens. Two patients were diabetic, 19 were hypertensive, and 20 were obese.^{13,16,18,19,27}

There was a prior history of intrauterine radium treatments for benign uterine bleeding in four other patients.

d. Symptoms

Because of earlier spotting, curettage had been performed previously for 19 of these patients (25.3%), from one month to seven years before this operation. Findings then had included endometrial polyps in 5, cystic hyperplasia in 11, and adenomatous hyperplasia in 3 patients.

Prolapse and vaginal relaxations were the only symptoms for 7 patients (see Table VI); after vaginal hysterectomy and plasty, microscopic sections disclosed adenomatous hyperplasia of the endometrium.

Spotting bleeding from the uterus was the primary symptom for 68 patients (90.7%). Ten of these patients also had heavy bleeding, and three had mild secondary anemia.

e. Microscopic Findings

Tissues from all 75 patients showed predominantly adenomatous hyperplasia of the endometrium. However, almost half of these specimens also included cystic hyperplasia and/or endometrial

polyps. Also, one-third of the patients also had notably atypical cells in adenomatous endometrial hyperplasia (Table V).

Moreover, portions of the endometria of 13 patients (17.3%) disclosed carcinoma of the endometrium (Table V).

Table V		
Microscopic Findings in Association with Adenomatous Hyperplasia of Endometrium (75 Patients)		
Diagnosis	Number	Percentage
Endometrial polyps	24	32%
Cystic hyperplasia of the endometrium	25	33%
Adenomatous hyperplasia of the endometrium	75	100%
Atypical adenomatous hyperplasia of the endometrium	19	25.3%
Carcinoma in situ of the endometrium	5	17.3%
Stage I a. carcinoma of the endometrium	3	
Stage I b. carcinoma of the endometrium	5	

f. Operative Procedures

All but 10 of the 75 patients who had adenomatous endometrial hyperplasia needed immediate or subsequent definitive operations (Table VI). Among those 10 (13.3%), there has not yet been any recurrence or development of other significant problems during the intervening 28 to two years (average 13.4 years)¹⁹.

Vaginal hysterectomy and plasty specifically treated the relaxations of seven otherwise symptomless patients; microscopic sections from these uteri disclosed adenomatous hyperplasia of the endometrium.

Table VI	
Operative Procedures 75 Patients with Adenomatous Endometrial Hyperplasia	
Procedures	Number
Dilatation & curettage, only	10
Vaginal hysterectomy	7
Dilatation & curettage, abdominal total hysterectomy, and bilateral salpingo-oophorectomy	30
Dilatation & curettage—later abdominal total hysterectomy and bilateral salpingo-oophorectomy, after an interval of one week to five years	28

In 30 other patients, the curettings were grossly typical of marked endometrial hyperplasia, or early

endometrial cancer. Additionally, these patients each had other significant abnormalities which are listed in Table VII.⁴⁴ Therefore, curettage was followed immediately by abdominal total hysterectomy (one radical), and bilateral salpingo-oophorectomy,

Table VII

Dilatation and Curettage and Immediate Abdominal Total Hysterectomy Plus Indicated Adnexa

Adenomatous Hyperplasia	30
Cervical Dysplasia	3
Cervical Carcinoma-in-situ	1
Cervical Cancer, Stage I	1
Adenomyosis	3
Myomas	9
Uterine Hypertrophy	4
Endometrial Cancer	13
Ovarian Neoplasia	3
Ovarian Cancer	1
Endometriosis	4

while using special cancer precautions during the operations (see Comments, concerning treatment).

In 28 other patients there was no urgent indication for hysterectomy at the time of curettage. However, within one month to five years, these 28 patients had developed the abnormalities listed in Table VIII.⁴⁴ Curettage, hysterectomy, and salpingo-oophorectomy were performed. Two patients had developed endometrial cancer during the intervening one, and five years, respectively. One patient had developed ovarian cancer, four years after prior curettage for adenomatous endometrial hyperplasia.

Table VIII

Dilatation and Curettage

Adenomatous Hyperplasia28
Delayed Abdominal Total Hysterectomy
Plus Indicated Adnexa

Uterine Prolapse	3
Vaginal Relaxations	6
Cervical Dysplasia	2
Cervical Carcinoma-in-situ	1
Adenomyosis	6
Myomas	9
Uterine Hypertrophy	4
Endometrial Cancer	2
Ovarian Neoplasia	5
Ovarian Cancer	1
Endometriosis	4

In summary, among 75 patients whose microscopic sections showed adenomatous endometrial hyperplasia, 13 had associated early endometrial cancer, and 2 subsequently developed endometrial cancer—a total of 15 patients (20%). Also, one patient had a granulosa cell cancer of the ovary, and

another patient subsequently developed a primary, epithelial cancer of the ovary. For many reasons, 65 of these patients needed to have uteri and indicated adnexa removed. Only 10 patients (13.3%) have not yet needed hysterectomy.¹³

4. Carcinoma of the Endometrium

Among the 6,491 patients who needed gynecologic operations, 84 (1.29%) were found to have carcinoma of the endometrium.^{12,39,45}

a. Follow-up

All of these patients have been evaluated by periodic examinations since their respective operations for endometrial cancer, during intervals of 1 to 30 years, until the present, or until death. Other medical conditions (Table IX) have caused later death of 29 of these patients (34.5%); each of these 29 patients lived between 3 and 25 years (average 11 years) after respective operations for endometrial cancer, reaching ages of 53 to 95 (average age of death, 76.2 years). Most notably, post-mortem examinations revealed that three patients had died respectively, secondary to (1) primary cancer of the pancreas, 5 years after endometrial cancer; (2) primary cancer of the sigmoid, 10 years after endometrial cancer; and (3) primary cancer of the gallbladder, 12 years after endometrial cancer.

Four patients have died secondary to or associated with metastatic endometrial cancer.

The remaining 51 patients are alive and healthy for intervals of 1 to 30 years after respective operations (average 12.56 years).

Table IX

Causes of Death of Patients Who Had Had Prior Endometrial Cancer

Causes of Death	Number of Patients
Cardiac conditions	14
Stroke	8
Diabetes	2
Renal failure	1
Fractured hip	1
Carcinoma, sigmoid	1
Carcinoma, pancreas	1
Carcinoma, gallbladder	1
	<u>29</u>

b. Age

Carcinoma of the endometrium was not found in any patient under age 40 (Table X). Conversely, endometrial cancer was diagnosed in 7 patients who were 80 or older (one was 86 years old). Most patients (70.2%) were between 45 and 70 years old.^{10,12,37,39,53,54}

The 86-year-old patient lived free of disease until age 92, at which time she died of "cardiac standstill." One 85-year-old patient lived for 10 years and died of cardiac failure at age 95. Two 80-year-old patients lived for 10 years and died of strokes at age 90. Apparently, advanced age is not a contraindication to adequate operative treatment for carcinoma of the endometrium.

Table X	
Age	
When Carcinoma of the Endometrium Was First Discovered	
Age Range	Number of Patients
40-49	15
50-59	23
60-69	26
70-79	13
80-86	7
	84

c. History

Among these 84 patients, 24 (28.6%) were nullipara, and 6 others had had only one pregnancy—a very low fertility rate for women in this region. Only 6 patients (7.14%) had any history of prior oral or injectable estrogens. However, another patient had been using large doses of ("Aristocort") cortisone for some years to treat rheumatoid arthritis, at the time her endometrial cancer was discovered. Ten patients were diabetic (11.9%), 36 patients were obese (42.8%), and 43 were hypertensive (51.2%).^{3,10,16,18,19,25,28,32}

Irradiation for treatment of benign bleeding had been administered earlier to 6 of these 84 patients (7.14%).

d. Symptoms

Because of earlier episodes of spotting, curettage had been performed previously for 38 of the 84 patients (45.24%). At that time sections had shown adenomatous hyperplasia of the endometrium for

six patients and endometrial polyps for 16. However, the previous microscopic diagnosis was not known for the remaining 16 patients.

Only one of these 84 patients had no spotting or discharge from the uterus when carcinoma was discovered. This patient had uterine descensus and vaginal relaxations, treated by vaginal hysterectomy and plasty; sections of her endometrium displayed "microfocal carcinoma," limited to portions of the endometrium.

Either postmenopausal or intermenstrual spotting bleeding from the uterus was the significant symptom of endometrial carcinoma for 78 (92.9%) of the remaining 83 patients. Three elderly patients had only a serous, pink discharge, and two elderly patients had only a purulent discharge from the uterus.

The duration of these symptoms (Table XI) before each patient sought medical care varied greatly. However, it was generally proportional to the depth of myometrial invasion of the endometrial cancer.^{6,33,37,39} The four patients who eventually died of endometrial cancer had had an average duration of **spotting bleeding** of approximately **80 months** prior to seeking definitive medical care.

e. Microscopic Findings

Tissues from these 84 patients were divided microscopically into well-differentiated, 41 patients; moderately-differentiated, 28 patients; poorly-differentiated, 15 patients.^{37,38,45,47}

Endometrial polyps were present in 16 patients (19%), and adenomatous hyperplasia was also present in 13 patients (15.5%). However, endometrial cancer had developed in otherwise atrophic endometrium in 19 older patients (22.6%). In endometrial tissues from the remaining 36 patients (42.9%), there were some elements of cystic hyperplasia.¹⁶

f. Staging

After careful study of (1) our drawings and descriptions in office records, (2) our operative re-

Table XI					
Duration of Spotting Bleeding Before Medical Care, Related to Depth of Myometrial Invasion (84 patients)					
Depth of Invasion		Number of Patients	Duration of Spotting (Months)		
			Least	Average	Most
Cancer in Myometrium	Cancer Limited to Endometrium	16	1	2	12
	Medial Third	37	1	6.73	48
	Middle Third	23	1	17.96	120
	Lateral Third	8	4	59.63	180

ports, and (3) individual microscopic findings, the staging of each of these 84 endometrial carcinomas was reevaluated. They are grouped as noted in Table XII.

Table XII

Stages
Endometrial Carcinoma
(84 Patients)

Stage	Number of Patients	Percent
0	6	7.15
I,a	35	41.67
I,b	26	30.95
II	8	9.52
III	8	9.52
IV	1	1.19

g. Depth of Myometrial Invasion

In this study, the depth of myometrial invasion (Table II) constituted the most effective criteria for evaluating duration and extent of disease^{6,9,14,33,37} (Table XI) and eventual prognosis^{6,9,10,33,37} (Table XIV).

h. Treatment

Generally, the key to successful treatment of endometrial cancer is prompt, meticulous, operative removal of the uterus, cervix, tubes, and ovaries.^{4,9,15,18,19,33,37,39,41,48} Technique should contain cancer, and avoid spillage or extrusion of cancer cells during the operation. In some patients various elements of radical hysterectomy may be needed.^{41,48} Some gynecologists prefer preoperative intrauterine radium or external irradiation. Others prefer not to irradiate some patients—particularly those having early, Stage I cancers^{6,9,14,18,33,37,38,41,48} (See Comments concerning treatment).

We have chosen to individualize treatment, based upon the age of the patient, her health, her sexual activity, and the extent of the disease (Table XIII). As a result, 24 patients have had operations alone. Only 8 patients have received both preoperative and postoperative irradiation. And, 52 patients have received operations, followed by external irradiation, which had been started during the first postoperative week. The very few extensions or recurrences of endometrial cancer (found after initial therapy) have been variously treated by (1) excision,^{7,18} (2) irradiation,^{14,18} and/or (3) large-dose progestin therapy.^{15,13,18,30,33}

i. Survival

Among the 84 patients who were treated for endometrial cancer, 4 patients (4.76%) died with known endometrial cancer (Figure 1, black dots). Three of these patients died within two years.^{10,15,20,32,36,51} The fourth patient's metastasis had

been treated for years by reoperation and progestin therapy, but she finally died 14 years after original diagnosis and therapy. The immediate causes of death were complications of hypertensive cardiovascular disease.^{15,20,32,36,51}

Table XIII

Methods of Treatment
For Endometrial Carcinoma
(84 Patients)

Abdominal Total Hysterectomy Bilateral Salpingo-oophorectomy	22
Abdominal Total Hysterectomy Bilateral Salpingo-oophorectomy Postoperative Irradiation	44
Abdominal Radical Hysterectomy Bilateral Salpingo-oophorectomy	2
Abdominal Radical Hysterectomy Bilateral Salpingo-oophorectomy Postoperative Irradiation	8
Preoperative Irradiation Abdominal Total Hysterectomy Bilateral Salpingo-oophorectomy Postoperative Irradiation	8

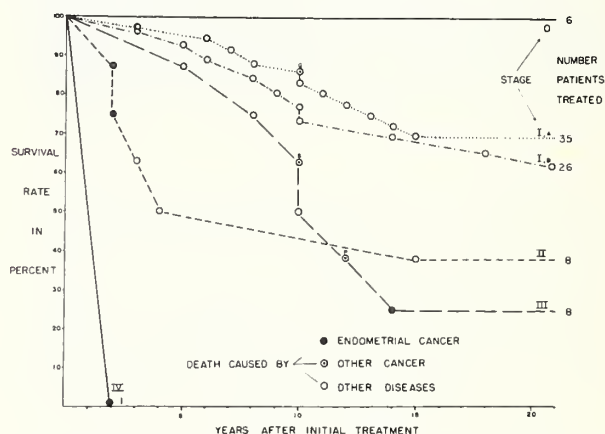


Figure 1

Long-term survival (all causes) after treatment for endometrial cancer.

Two other patients had known recurrences of endometrial cancer. In one patient, two years postoperatively, a small vaginal recurrence was excised, and the pelvis was treated by external irradiation. The patient has no known cancer now, five years postoperatively. Another patient had an isolated liver metastasis excised surgically four years after her initial cancer operation; she died of cancer of the pancreas 12 years after her initial therapy for endometrial carcinoma.

Therefore, 78 patients (92.86%) have survived, apparently free from endometrial carcinoma since their original treatments, and 80 patients (95.24%) have survived, apparently free of endometrial can-

Table XIV											
Prognosis											
4 Patients Who Died with Endometrial Cancer											
Evaluation at Time of Diagnosis											
Endometrial Cancer Was First Treated	Gross Stage				Micros. Different			Depth of Myometrial Invasion			
	0 I,a I,b	II	III	IV	G ₁	G ₂	G ₃	none	medial	middle	lateral
1954			X			X					X
1962		X					X				X
1964		X					X				X
1968				X		X					X

cer after primary and secondary treatment. If one eliminates the six patients with Stage 0 carcinoma ("endometrial carcinoma in situ") from the computations,¹⁹ these overall survival percentages, free of endometrial cancer, are reduced respectively to 92.3% and 94.9%.

It is not always easy for pathologists to identify, during autopsy, the exact origin of adenocarcinoma. Therefore, if one liberally includes, among the endometrial cancer deaths, the three patients who died of primary cancer of the pancreas, gallbladder, or sigmoid, 6 to 12 years after operation for cancer of the endometrium, then the overall survival rate, free of cancer, is 91.66%.

No patient with Stage I disease has died from endometrial carcinoma. There were 17 patients who had Stage II, III, or IV cancer; the survival rate for these 17 patients was 76.5%.

No patient has died of endometrial cancer who had her initial operation within the past 11 years.

Since 20 of these patients were 70 or older when endometrial cancer was first discovered and treated, one could anticipate that many deaths from other causes would occur within the next two decades.²⁰ If one subtracts the deaths caused directly by cancer, it is interesting that long-term attrition, due to other diseases, was increased significantly among the patients who had advanced stages of endometrial cancer (about 42% for combined Stages I.b, II, III, and IV), compared to patients with earlier cancer (about 27% for Stages 0 and I.a). This perceptible difference in long-term survival may have been caused by secondary stresses on body physiology, resulting from the more extensive operations, and particularly the irradiations which were received by patients who had Stage I.b, II, III, or IV cancer.^{9,10,37,41}

j. Prognosis

In this study, gross staging and the degree of

microscopic differentiation were only moderately helpful in predicting long-term results. The depth of myometrial invasion was more helpful (Table XIV).^{6,9,10,33,37} Myometria were infiltrated by cancer to the lateral third (almost to the serosa) in all four patients who subsequently died with endometrial cancer. No lymph glands were palpable in one of these patients.

Four other patients also had endometrial cancer invading the lateral third of the myometrium; 3 lived 5 to 15 years, after operation and irradiation, without evidence of cancer, and died of other medical conditions; one is alive and well 5 years after operation and irradiation.^{9,10,20,33,37}

Because of the length of this study, COMMENTS concerning its interrelations will be published in the **SOUTH DAKOTA JOURNAL OF MEDICINE** next month under the following headings:

1. Relationship Between Types of Endometrial Neoplasia.
2. Potential Etiologic Factors; History and Symptoms.
3. Diagnosis and Treatment of Endometrial Cancer and Precursor Lesions.
4. Survival and Prognosis.

PROMOTING YOUR BELIEFS SODAPAC

If your practice is Incorporated, SoDoPAC and AMPAC voluntary political contributions should be written on a PERSONAL CHECK. Contributions are not limited to the suggested amount. Neither the AMA nor the SDSMA will favor or disadvantage anyone based upon the amounts of or failure to make PAC contributions. Contributions are subject to the limitations of FEC Regulations, Sections 110.1, 110.2 and 110.5 (Federal regulations require this notice). Copies of SoDoPAC and AMPAC reports are filed with the Federal Election Commission and are available for purchase from the Federal Election Commission, Washington, D.C.

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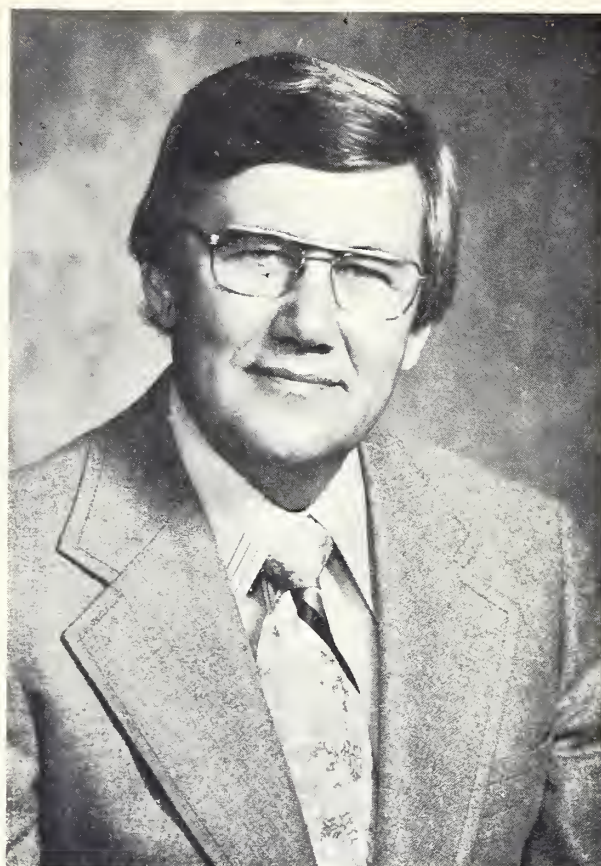
President's Page

A recent malpractice claim was filed against a physician when it was ruled that a child was permitted to sue a physician who had treated her mother when she herself was a child—a full nine years **before** the daughter plaintiff was born. Another recent case awarded a woman \$800,000 because, following surgery, her “belly button” was off center.

The above examples are a warning to us all that the professional liability crisis again is rearing its ugly head, both because of unusual suits as well as extremely high payouts. St. Paul Fire and Marine has released some sobering figures that show claims which had decreased as much as 11% in 1976, were up 12% in 1978, and awards by the court were up as much as 100% in some states since 1976. In our own state, somewhere in the neighborhood of 115 allegations are now on file with St. Paul. Many of these are without any merit and most will be resolved with no damages allowed against the physician involved. However, almost all of these cases will cost something to mediate or defend, and so our professional liability insurance will again increase.

We can criticize the suit conscious public, who seem to insist on perfect results and solution, or we can villify those blankity-bleep lawyers and their contingency fees and assume they are the cause, being nothing but a bunch of ambulance chasers. Perhaps there is some truth to the above assumptions, but I think it is time for all of us to do a little soul-searching and re-evaluation of our medical practices that relate to possible litigation.

St. Paul lawyers, who defend many physicians in our state, have found a startling trend in many suits filed in South Dakota. More suits are instigated due to the fact that one physician criticizes a physician or insinuates that the previous physician treated the patient improperly. I would never suggest that we condone poor treatment that injures a patient. However, it is unconscionable, and perhaps unethical,



to criticize a fellow physician's treatment without first ascertaining all of the facts.

There are so many legal pitfalls in the practice of medicine, it would be unending to enumerate them, but we must continue to practice sound, up-to-date medicine. We must realize it is impossible to know everything in all fields, and when a medical or surgical problem arises that may be too intricate for our skills, to ask for help from someone with a little more expertise. Our ego must not be satisfied at the expense of our patients, lest we find ourselves before a judge and jury.

South Dakota physicians are as dedicated, as skilled, and as kind as anywhere in this country. Yet all of us can stand to be reminded to treat the patient with sympathy and listen with patience and concern, particularly when a bad result, either real or imagined, occurs. The practice of medicine is terribly complex, but if we truly avoid its many legal pitfalls, we can help our lawyer friends find more time to prepare deeds, wills, agreements, etc.

Sincerely,

D. B. Reaney, M.D., President
South Dakota State Medical Association

5th ANNUAL BLACK HILLS WINTER SKI SEMINAR

ON DIABETES MELLITUS, GASTROENTEROLOGY, OBESITY, PREVENTIVE MEDICINE

JANUARY 31, FEBRUARY 1, 2, 1980

NORTHERN BLACK HILLS HOLIDAY INN & SOLAR DOME, SPEARFISH, SOUTH DAKOTA

Hosted By: South Dakota Chapter of the American Academy of Family Physicians

This program is acceptable for 12 prescribed hours by the American Academy of Family Physicians and 12 hours Category 1, AMA credits.

WEDNESDAY, JANUARY 30, 1980

7:00-9:00 p.m. Registration
7:00 p.m. SDAFP Board of Directors Meeting — Deer Mountain Room

7:30-8:10 a.m. "Potential for Prevention of CHD by Multirisk Factors"
Arthur M. Leon, M.D.
8:15-8:55 a.m. "Causes of Obesity"
Gloria Leon, Ph.D.
9:00-9:40 a.m. "Acute and Chronic Effects of Exercise Training on the Body"
Arthur M. Leon, M.D.
9:45-5:30 p.m. WINTER SPORTS TIME

THURSDAY, JANUARY 31, 1980

MORNING SESSION

William Tschetter, M.D., Moderator

7:00-7:30 a.m. Registration, Complimentary Continental Breakfast
7:30-8:10 a.m. "Who Gets Gallstones and Why"
Robert R. Raszkowski, M.D.
8:15-8:55 a.m. "Inflammatory Bowel Disease, Diagnosis and Treatment"
Kenneth Vogele, M.D.
9:00-9:40 a.m. "So Your Patient Has Gallstones"
Robert R. Raszkowski, M.D.
9:45-5:30 p.m. WINTER SPORTS TIME

EVENING SESSION

Gary Welsh, M.D., Moderator

5:30-6:10 p.m. "The Loose Goose — Basic Diarrhea Workup"
Kenneth Vogele, M.D.
6:15-6:55 p.m. "The Albatros Syndrome"
Robert R. Raszkowski, M.D.
7:00-7:40 p.m. "Troubleshooting Common GI Problems"
Kenneth Vogele, M.D.
7:45-9:00 p.m. Complimentary hot wine and hot buttered rum — Poolside

FRIDAY, FEBRUARY 1, 1980

MORNING SESSION

Lawrence W. Finney, M.D., Moderator

7:00-7:30 a.m. Registration, Complimentary Continental Breakfast

EVENING SESSION

Michael Brown, M.D., Moderator

5:30-6:10 p.m. "Prevention of Eating Disorders"
Gloria Leon, Ph.D.
6:15-6:55 p.m. "Clinical and Diagnostic Uses of Insulin"
Fred C. Lovrien, M.D.
7:00-7:40 p.m. "Evaluation of Current Treatment Methods of Obesity, with Detailed Discussion of Behavior Modification Techniques"
Gloria Leon, Ph.D.
7:45-9:00 p.m. Cash bar and complimentary hors d'oeuvres — Poolside

SATURDAY, FEBRUARY 2, 1980

MORNING SESSION

Daniel Sherry, M.D., Moderator

7:00-7:30 a.m. Registration, Complimentary Continental Breakfast
7:30-8:10 a.m. "Diabetic Ketoacidosis"
Fred Lovrien, M.D.
8:15-8:55 a.m. "Exercise and Diet Prevention and Control of Adult Onset Diabetes Mellitus"
Arthur M. Leon, M.D.
9:00-9:40 a.m. "Recent Advances in Diabetes Mellitus"
Fred C. Lovrien, M.D.
9:45 a.m. Seminar Closes

**MAKE PLANS TO ATTEND NOW. WRITE:
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SD

Chapter News



SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS
3001 South Holly Avenue
Sioux Falls, SD 57105



Results Of Annual Survey Of Family Practice Residency Programs August, 1979

I. Programs:

A. Total Approval Programs		364
B. Total Operating Programs (9 approved but not operating)		355
Community Hospital Based	57	
University Based	62	
University Affiliated or Administered	228	
Military Hospital Based	17	

II. Residents:

A. Total Residents			6,531
1. Total First Year Residents	2,360		
2. Total Second Year Residents	2,205		
3. Total Third Year Residents	1,966		
B. Total Approved First-Year Positions			2,500
C. First Year Fill Rate			94.4%
D. Increase/Decrease Class Size by Year			
	1977-78	1978-79	1979-80
Class of '80	2,043	1,986	1,966
Class of '81	—	2,318	2,205
Class of '82	—	—	2,360

III. Residency Graduates:

A. Total July, 1979 residency graduates	1,724
B. Total graduates from family practice residency programs since January 1, 1970	6,666



Governor Janklow Wins DSA Plaque

Governor William Janklow was presented with the first SDAFP Distinguished Public Service Award during the 1979 Black Hills Summer Seminar in Spearfish. The Governor was given this award for his efforts in promoting and supporting family practice in South Dakota.

Left to Right, Richard Friess, M.D., R. G. Nemer, M.D., James Ryan, M.D., Governor Bill Janklow, and B. O. Lindbloom, M.D.

SD Laboratory Aids

Sponsored by the South Dakota Society of Pathologists

Pheresis—A Technique Come of Age

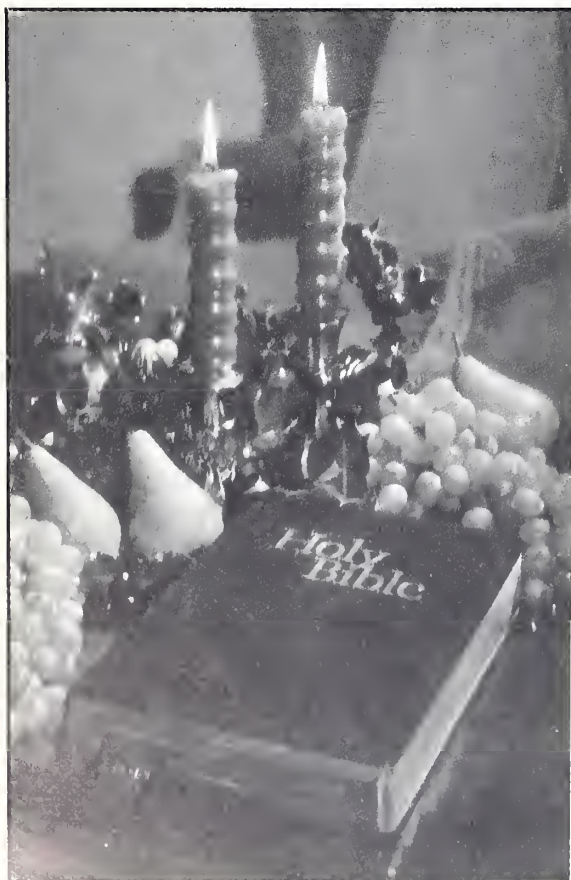
Pheresis refers to removal of blood, the separation and acquisition of a single blood component, and return of the remainder of the blood to the original donor. The component removed from the donor is indicated by the name of the procedure—plasmapheresis (removal of plasma), plateletpheresis (removal of platelets), leukapheresis (removal of white cells) etc. The technique is usually accomplished by continuous or discontinuous centrifugation.

The major stimulus for pheresis procedures has been the advent of cancer chemotherapy resulting in marrow depression. Red cell deficits can easily be remedied by transfusions of a small number of units every few weeks or months. Leukocytes, however, are present in only relatively small quantities in a single unit of blood and leukocyte survival is

only a matter of hours in the circulation. To a lesser extent the same can be said for platelets which survive for over a week. Also long term storage of either leukocytes or platelets is not yet available at the community hospital level. In order to obtain the required number of white cells and/or platelets for a cytopenic patient from a donor, several units of blood are circulated through a centrifugal instrument and the cells are collected as a concentrate which must be transfused within a 24 hour period.

Platelets may be given as needed every few days or weeks. Leukocytes are usually given in courses one each day for four to six days. This type of therapy is usually reserved for infected (white cells) patients or bleeding (platelets) patients during intensive periods of chemotherapy.

John F. Barlow, M.D.
Pathologist



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Thank you for making this territory one of the most successful for the Dow Chemical Company during the past twenty-eight years.



R. L. "Russ" Bonacker
Medical Service Representative
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Fifteen Year Old Male and Six Month Old Male With Marked Hypoproteinemia and Edema

Kurt E. Johnson, M.D.*

John D. Barker, M.D., FACP**

Discussers

John F. Barlow, M.D., FCAP***

Editor

Case #787735

This six-month old male infant was sent to a pediatrician for evaluation because of purple feet and a rash on the face. The patient was seen approximately six weeks prior to admission for the above complaint associated with anorexia. The patient seemed to be getting progressively weaker, stopped smiling and acted like he did not feel well. Because of increasing edema of the lower extremities, he had an outpatient 12 panel examination which revealed a very low protein value. Urinalysis at that time was negative. The patient had weighed 7 lbs. 4 oz. at birth. There were no complications in the prenatal or postnatal course. The patient had developed normally until the present illness. The patient had breast fed normally and almost exclusively with exception of a few soft foods. There was no family history of metabolic disorders within the family. The patient had a 4 year old sister who was normal.

PHYSICAL EXAMINATION: Temperature 99°F, pulse 140/minute and regular; respirations 52/minute and regular, blood pressure 90 systolic and 60 diastolic, height 65.8 cms., and weight 16 lbs. ½ oz. Head circumference 43 cms. The patient was lethargic but had no abnormalities of the head or neck. The lungs were clear to auscultation and percussion. The heart was not enlarged and there

were no abnormal sounds. Examination of the abdomen revealed no palpable organs, masses, or tenderness. There was marked edema with some pitting of the lower extremities and slight cyanosis of the feet. The arms showed mild edema which was non-pitting. There was a doughy appearance to the abdomen and the skin. There was a rash over the cheeks. Neurologic examination was within normal limits.

LABORATORY DATA: Urinalysis yellow, clear; specific gravity 1.007; pH 5.5, negative for protein, reducing substances, glucose, ketone bodies, bile and hemoglobin. There was a trace hemoglobin on one occasion on several urinalyses, but others were negative. The sediment of the urine showed 1-4 white cells, 0-1 red cells/hpf on several specimens with 3-5 hyaline casts on one occasion. Glucose and protein determinations were repeated multiple times in the urine and showed no higher readings than 1+ on either determination. Hemoglobin 17.3 gm/dl, hematocrit 46 vol./dl, normal indices, total leukocyte count 14,400/mm³ with 78% segmented neutrophils, 10% neutrophilic bands, 2% eosinophils, and 10% lymphocytes. The red cells were normochromic and normocytic with slight anisocytosis and the platelets were normal in number and morphology. Zetacrit was 48% (normal). 12 panel showed a lactic dehydrogenase which was markedly elevated (hemolytic specimen). Alkaline phosphatase was 143 IU/L (normal for age). Serum aspartate aminotransferase was 320 IU/L (normal 0-100 IU/L); calcium 7.3 mg/dl (normal 8.4-10.7 mg/dl); total protein 2.2 gm/dl (normal 8.4-10.7 mg/dl); total protein 2.2 gm/dl (normal 5.5-8.3 gm/dl). Total bilirubin, inorganic phosphorus, glucose, blood urea nitrogen, creatinine, uric acid were within normal limits; cholesterol was 100 mg/dl; one stool guaiac was positive for occult blood. Electrolytes revealed pH 7.26, PCO₂ 34 torr; bicarbonate 16 mm/L; sodium 124 meq/L; potassium 3.0 meq/L and chloride 101 meq/L.

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***Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital, Sioux Falls, SD; Professor of Pathology, School of Medicine, University of South Dakota.

Stool culture and stool for ova and parasites were negative. D-xylose absorption test was within normal limits. Stool for reducing substance was within normal limits. Serum protein electrophoresis albumin 1.4 gm/L, alpha I globulin 0.2 gm/L; alpha II globulin 0.3 gm/L; beta globulin 0.26 gm/L, gamma globulin 0.4 gm/L, total protein 2.3 gm/L, prothrombin time 16 seconds with a control of 12 seconds, partial thromboplastin time was 167 seconds with a control of 35 seconds. A chromium 51 labelled albumin study showed markedly elevated levels (12% of the administered dose in the stool compared to the normal less of 0.7%). Serum zinc and VMA studies on the 24 hour urine were within normal limits; magnesium fluctuated from levels below 1.0 meq/L to within normal range of 1.5 to 1.9 meq/L.

The patient's course was characterized by frequent loose mushy or watery stools which were bright yellow, hypoproteinemia, hypokalemia, and slightly abnormal liver function tests but a liver spleen scan was within normal limits. X-rays of colon and upper gastrointestinal studies, including a small bowel follow-through were within normal limits. Chest film was unremarkable except for a question of peri-hilar inflammation on one reading. An electrocardiogram revealed sinus tachycardia. The patient had one bout of respiratory difficulty requiring intubation and ventilator support. However, the patient was rapidly weaned from the respirator. The patient was treated with hyperalimentation of 30 mg/br. including 14 gms. of amino acid, 40 meq. of potassium; 24 meq. of calcium. He was also given 7 gm. of albumin intravenously every six hours and 1 ml. of magnesium sulfate and vitamin K as needed. The patient was transferred for further evaluation after a capsule biopsy of the small intestine failed to obtain a specimen.

Case #805416

This 15-year-old male was admitted to Soux Valley Hospital with chief complaint of diarrhea of six weeks duration.

The child had been born with cyanotic congenital heart disease and four years prior to admission had had a Fontan procedure which was the formation of a conduit from the right atrium to the right infundibulum for tricuspid atresia with atrial septal defect and ventricular septal defect and persistent left superior vena cava. The patient had been given digoxin and spironolactone after surgery. The patient had had some infraorbital edema and itchy eyes with tearing since the operation. Because of the infraorbital swelling, the patient was started on a thiazide diuretic. The patient also had a history of two weeks of left-sided pressure in the chest with some numbness of the left lateral arm. There was no shortness of breath, diaphoresis, vomiting, abdominal pain or melena. The stools were gray-brown in color. There was no family history of diarrhea, but there was a family history of allergy.

PHYSICAL EXAMINATION: Temperature 98.6°F; pulse, 80/minute and regular; respirations: 18/minute and regular; blood pressure 90 systolic and 56 diastolic; height 4'8"; weight 82½ lbs. Examination of the head and neck was unremarkable. The lungs were clear to auscultation and percussion. The heart showed a normal sinus rhythm with no abnormal sounds. The abdomen was soft without palpable tenderness, organs, or masses. Neurological examination was within normal limits. There was no cyanosis or clubbing. Genitalia were normal for age.

LABORATORY DATA: Urinalysis; straw-colored, clear, specific gravity 1.019; pH 5.0, negative for protein, glucose, ketone, bile and hemoglobin; sediment negative, hgb. 13.7 gm/dl with normal indices. White count 6,800/mm³

with 84% segmented neutrophils, 3% neutrophilic bands, 4% eosinophils and 9% lymphocytes. The red cells were normochromic and normocytic and the platelets normal in number and morphology. pH 7.34, pCO₂ 36 torr, bicarb 25 mm/L, sodium 131 meq/L, potassium 2.9 meq/L, chloride 103 mg/L. Total protein 3.0 gm/dl with 1.5 gm/dl, albumin and 0.3 gm/L alpha I globulin 0.6 gm/L, alpha II globulin, 0.5 gm/L beta globulin and 0.1 mg/L gamma globulin, (the albumin and gamma globulin were markedly depressed). Lactic dehydrogenase, alkaline phosphatase, aspartate aminotransferase, total bilirubin, inorganic phosphorus, glucose, blood urea nitrogen, creatinine, and cholesterol were within normal limits. Uric acid was 2.2 mg/dl, calcium 6.9 mg/dl, and total protein 2.9 mg/dl. A fluorescent antinuclear antibody test (FANA) was negative. IgG was 160 mg/dl (normal 750-2000 mg/dl) IgM 42 mg/dl (normal 63-250 mg/dl) IgA 60 mg/dl (normal 84-483 mg/dl) D-xylose showed normal plasma values at 34 mg/dl at 30 minutes and 84 mg/dl 1 hour (normal 30-40 mg/dl), total D-xylose 3.8 gms. excreted (normal 4.1 to 8.2 gms. excreted). A stool culture examination for ova and parasites was negative. Qualitative study for split and neutral fat were elevated in the stool. Febrile agglutinins for salmonella, brucella and tularemia revealed no titer. Electrocardiogram revealed anterior fascicular block and non-specific T wave abnormalities with an abnormal atrial focus. Chest film showed clear lungs with slight cardiomegaly. An upper gastrointestinal series was unremarkable, but there was prominence of folds throughout the small bowel. Serum carotene was 88 mcg/dl (normal 70-200 mcg/dl). A serum dioxin showed undetectable levels. Small bowel fluid was examined for *Giardia Lambia* at the time of intestinal biopsy and was negative. Small bowel biopsy was diagnostic.

DR. JOHNSON: I would like to begin this discussion by highlighting the parameters these two cases share in common—their young age, edema, diarrhea, marked lymphocytopenia, and hypoproteinemia. Striking hypoproteinemia in the face of normal hepatic and renal function should lead the clinician to consider other sources of protein loss. Both of these patients exhibited diarrhea; and, indeed, the gastrointestinal (GI) tract may be a source of protein loss. In normal individuals 10% of protein degradation can be attributed to enteric losses. The liver which on average produces 16-18 grams of albumin a day can increase its production two fold before it reaches maximal output. Normally the GI tract can breakdown proteins and absorb the constituent amino acids. If the mucosa is severely damaged by disease processes, it may lose its ability to perform this function. Similarly, diseases of the lymphatic system may be the etiology of GI protein losses.

A number of disease entities may be responsible for gastrointestinal loss of proteins. The term "protein losing enteropathy" has been applied to them. They may be grouped in three categories: 1) those with mucosal ulcerations, 2) those with impaired metabolism or increased turnover of epithelial cells, and 3) those with obstruction of intestinal lymph flow.

Gastric carcinoma, gastric lymphoma, multiple gastric ulcers, colonic cancer, and granulomatous enteritis are examples of diseases with mucosal ulceration that can lead to enteric losses of protein. Rugal hypertrophy (Menetrier's disease), celiac sprue, tropical sprue, Whipple's disease, allergic gastroenteropathy, bacterial and parasitic enteritis are examples of diseases without mucosal ulceration. Primary lymphangiectasia, lymphoma, and lymphenteric fistulas constitute diseases in which lymphatic abnormalities are responsible for protein loss.

Diagnosis of a protein losing enteropathy is established clinically by use of albumin labelled with chromium 51. The intravenously injected albumin is lost into the gut and can be measured in the stool. Normal values are less than 1%. The 6 month old infant showed a marked elevation in chromium labelled albumin in the stool - 12%, establishing GI loss as the source of this patient's severe hypoproteinemia.

Of the diseases that can cause protein losing enteropathy, intestinal lymphangiectasia seems to be the most likely cause in these two cases. Intestinal lymphangiectasia is a disorder of the lymphatic system characterized by peripheral edema, hypoproteinemia, lymphocytopenia, and gastrointestinal symptoms. The lymphatic vessels are dilated and telangiectatic in the small intestine and frequently hypoplastic in the extremities. This hypoplasia of the lymphatic vessels is thought to be the cause of the asymmetric edema seen in some patients.

The etiology of intestinal lymphangiectasia remains obscure. Congenital malformation is likely, especially in patients afflicted at an early age. If the symptoms appear later in life, frequently another disease process is responsible, and the lymphangiectasia appears to be an acquired defect. A host of diseases have been linked to intestinal lymphangiectasia. Retroperitoneal fibrosis, intestinal scleroderma, pancreatitis, constrictive pericarditis, congestive heart failure, regional enteritis, radiation enteritis, lymphomas, and abdominal tuberculosis have been incriminated. The pathogenesis in these cases is thought to be increased lymphatic pressure, leading to dilatation of the vessels and transudation of lymph into the intestinal lumen.

The congenital form of intestinal lymphangiectasia has no sex predelection and manifests itself before the age of 30. The edema as mentioned earlier may be asymmetrical and macular edema leading to reversible blindness has been reported. Typically the GI symptoms are mild with intermittent diarrhea and steatorrhea. If untreated, growth retardation can occur.

Serum protein studies show marked decreases in

albumin, IgA, IgG, and IgM. Lymphocytopenia is a universal finding, secondary to the loss of lymphatic fluid into the GI tract. Humoral immunity remains intact, but patients exhibit defective cellular immunity as evidenced by negative reactions to skin tests and failure to reject skin grafts. Radiologic findings are variable, frequently they are normal; however, edema of the bowel wall, thickening of the mucosal folds, and segmentation and dilatation of the bowel wall have been described. (Fig. 1)



Figure 1
Note thickened mucosal folds in small bowel.

Treatment consists of limiting dietary intake of fats and substituting medium chain triglycerides (MCT) for long chain triglycerides (LCT). Since LCT are transported by the intestinal lymphatic system to the thoracic duct, they increase intralymphatic pressure resulting in lymph leaking into the intestinal lumen. MCT are transported via the portal system, thus bypassing the already overloaded lymphatic system. Treatment of cardiac patients should be aimed at decreasing central venous pressure. Correcting their right heart failure, constricting pericarditis, or tricuspid regurgitation will lead to a lowering of central venous pressure, lessen lymphatic congestion, and decrease loss of lymphatic fluid into the intestinal lumen. Prognosis for patients with intestinal lymphangiectasia is good, if proper medical management is instigated.

Dr. Kurt Johnson's Diagnosis **Lymphangiectasia of the Small Bowel**

DR. BARLOW: The small bowel biopsy was an excellent specimen which had previously been well oriented by Dr. Barker. Flattening the biopsy on a piece of gauze or gelfoam is very important because

proper sections at right angles to the mucosa are critical in evaluating mucosal lesions of the small bowel. The first biopsy I am going to show is from the 15-year-old boy. A small bowel biopsy from the young child shows similar changes with dilated lymphatics but they are less dramatic than in the biopsy I will show. The first low power slide (Fig. 2) reveals that the villous architecture is intact but the tops of the villi appear clubbed and have central clearing. Higher power shows this central clearing is due to dilated spaces lined by endothelial cells which are typical of lymphatics. (Fig. 3) The epithelium is intact and there is a very mild chronic inflammatory infiltrate in the lamina propria of the small bowel. The pattern is diagnostic for intestinal lymphangiectasia.

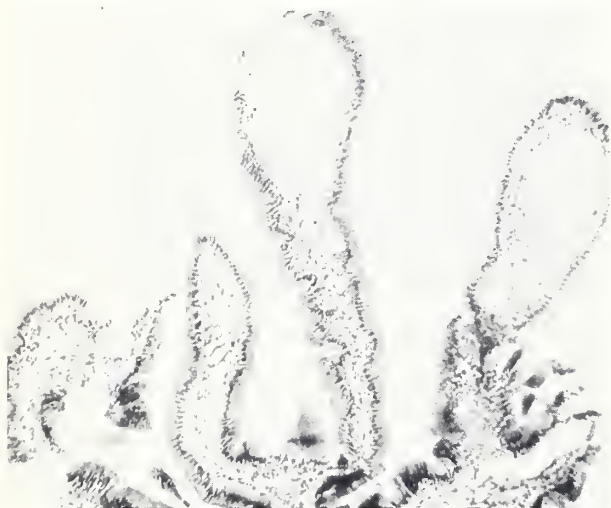


Figure 2
Dilated lymphatics in tips of villi (100 X).

DR. BARKER: That was an excellent summary of the entity of lymphangiectasia of the bowel and I will only review some significant aspects of these two cases. The young child is a classic case of congenital lymphangiectasia. The child had total body edema or anasarca. The original small bowel series on this patient was reported as normal. This is not an unusual circumstance in congenital lymphangiectasia. In fact, the lymphangiectasia may be very focal in this disease. If the patient is refractory to medical therapy, surgeons have attempted to operate on these patients by selecting the more severely affected segments utilizing an injection of dye during the operative procedure. It is sometimes possible to resect the most severely involved areas. One may obtain at least some degree of clinical decrease in severity of the gastrointestinal protein loss in this

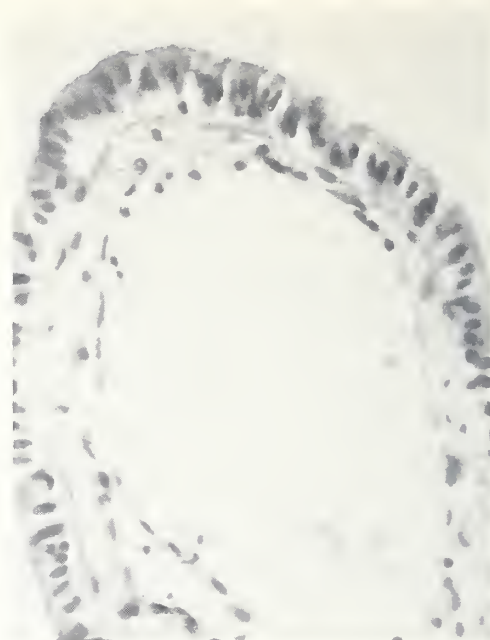


Figure 3
Closeup of villus showing dilated lymphatic with endothelial lining cells (430 X).

manner. It must be emphasized that because of the patchy nature of the disease, small bowel biopsies may be normal in these patients.

The total lymphatic system is totally deranged in these children. On lymphangiogram you can demonstrate ectasia, sclerosis, and occasional transudation of the radio-contrast material into the lumen of the bowel in these patients. In one case reported at a conference the massive protein loss in the stool caused yellow stools in a woman. They had injected a blue dye in the lymphangiogram and subsequent stools were colored blue. However, before a picture could be taken, someone shook the container of feces and everything turned green.

The second case of the teenage boy is particularly interesting because I feel that the procedure performed for the correction of the congenital heart disease precipitated the lymphangiectasia of the bowel with protein loss. At age 12 or three years before this admission, the patient had had a Fontan procedure for hypoplasia of the right heart. The cardiac anomaly included tricuspid valve atresia, atrial septal defect and ventricular septal defect. The aim of the Fontan correction procedure was to bypass the hypoplastic right heart by diverting blood from the right atrium into the pulmonary circulation by a shunt. The procedure was very successful and the patient's exercise tolerance improved greatly. However, a year after the operation the patient

noted that one side of his face was swollen when he woke up in the morning. The edema regressed as the day passed on and the mother attributed the edema to allergy. A likely explanation for the patient's symptoms is that when the blood from the superior vena cava was diverted into the pulmonary artery circulation and the septal defects were closed, there was a marked increase in pressure in the superior vena cava. Since the thoracic duct empties into the junction of the left internal jugular and subclavian vein and there was marked increased pressure postoperatively in the lymphatic system. This caused dilated lymphatics in the bowel. The lymphangiectasia in this case was due to the pressure dynamics in the cardiovascular system and it is a good example of acquired lymphangiectasia. A similar sequence of events probably occurs in lymphangiectasia of the bowel seen secondary to prolonged congestive heart failure or constrictive pericarditis. Not until the patient had diarrhea and signs of protein-losing enteropathy was the connection with the cardiac operation suspected. In discussing this case with the pediatric cardiologist in Minnsota, this has been seen before after this operation. A similar syndrome has been reported after a mustard procedure in an attempt to correct transposition of the great vessels and after the establishment of a Glenn shunt for repair of a truncus arteriosus. This patient has been started on a diet of decreased LCT and increased MCT. This is the therapy that Dr. Johnson mentioned. The therapy will be only a palliative measure and I would expect the patient may get worse. The question is whether a cardiac procedure would be indicated to decrease the pressure on the lymphatic system. I feel the prognosis is not very good. It should be remembered that therapy with MCT is simply a method of allowing the patient to absorb fat. It allows one to decrease the amount of LCT in the diet. The decrease in the LCT in the diet tends to decrease the pressure in the lymphatic system of the bowel. Hopefully, this will decrease the amount of protein exudation into the bowel lumen. The MCT therapy does not improve other types of fat absorption and you must remember that the patient may become deficient in certain fat soluble vitamins.

The distinction between maldigestion as in pancreatic disease and malabsorption as in diseases

such as sprue is frequently made when discussing small bowel disease. However, here we have a third category of disease in lymphangiectasia. The patient has protein-losing enteropathy but he does not necessarily have steatorrhea. Some patients with protein-losing enteropathy may have no diarrhea or any bowel complaints at all. The 72-hour fat collection in this child was only mildly abnormal in the face of the marked protein loss.

Dr. Johnson mentioned that you get protein-losing enteropathy in regional enteritis. This can be due to mucosal ulceration but it often also is due to very extensive lymphatic obstruction in the submucosa.

I would like to point out that in retrospect, the small bowel x-rays in the older patient are abnormal since you can see very prominent folds in the small bowel. (Fig. 3) The x-ray appearance corresponds to the dilated lymphatics which have previously been shown on the biopsy.

*DR. C. D. SULLIVAN: Am I correct in stating that the loss of protein is simply due to increased pressure and when one injects the radioactive chromium, it simply diffuses into the bowel lumen?

DR. BARLOW: That is the way I understand it.

DR. JOHNSON: I should point out that lymphedema can lead to protein loss into extremities in a patient due to the hypoplasia of the lymphatics as well as exudation from the bowel.

There is another category of lymphangiectasia that has been described recently in the *March New England Journal of Medicine*.¹² They report three cases of what they call lymphangiectasia due to inflammation. The patients were all under 30 and had mild diarrhea and marked hypoproteinemia but normal or elevated immunoglobulin levels. Radioactive chromium-labeled albumin studies demonstrated loss of protein from the bowel. Lymphangiectasia was noted on small bowel biopsies. These patients had no kidney, liver or heart disease. Diagnostic laparotomy except for the lymphangiectasia of the small bowel revealed no significant findings. The patients all had elevated erythrocyte sedimentation rates ranging between 80 and 120 mm/hr. Corticosteroids seemed to alleviate the symptoms. The authors postulated a new type of lymphangiectasia due to inflammation.

**DR. P. K. ASPAAS: Is the marked hypocalcemia due to the markedly decreased protein only?

DR. JOHNSON: The low calcium is largely due to the low protein but some patients have been described with tetany so I suspect that decreased ionized calcium may occur.

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DR. ASPAAS: Is there any association with the congenital form of lymphangiectasia and so-called Milroy's disease or yellow nail syndrome?

DR. BARKER: I believe that congenital lymphangiectasia of the extremities has been called Milroy's disease. Protein loss into a lymphedematous extremities can occur⁸ but there is not necessarily an accompanying intestinal lymphangiectasia. I know of no association between the so-called yellow nail syndrome (lymphedema, pleural effusions and yellow-nails) and intestinal lymphangiectasia.

DR. SULLIVAN: Since this patient has swollen abnormal villi, is it possible that malabsorption of fat and other substances may become worse?

DR. BARKER: I suspect that indeed he may have malabsorption of other substances in the future; although, as I have noted, the fat absorption at this point is only mildly abnormal. Both patients do not have a good prognosis; and, as yet, we have no really effective therapy for their condition.

It should be noted that these patients do develop marked depression of the immunoglobulins as well as lymphocytopenia. Although this is true, they do not apparently develop serious infections because humoral immunity is intact. They do have impaired cellular immunity as evidenced by decreased ability to reject skin grafts and negative skin tests to certain antigens.

DR. BARLOW: There are a variety of types of primary lymphedema which usually involve the extremities. These can be due to aplasia, hypoplasia or hyperplasia of the lymphatic system. The hypoplastic type may be divided into obstructive and non-obstructive types. Milroy's disease is familial and often congenital, but may not be manifest until puberty (praecox form) or even after 35 years of age (Tarda form). Milroy's disease is probably of dominant inheritance. It is more common in females. Primary lymphedema is also associated with ovarian dysgenesis. Congenital lymphedema may or may not be associated with lymphangiectasia of the bowel. Protein loss into a lymphedematous extremity does occur.

FINAL ANATOMIC DIAGNOSES LYMPHANGIECTASIA OF THE BOWEL

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GREETINGS

FROM



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S D *Council Meeting Highlights*

The Council of the South Dakota State Medical Association met on Friday and Saturday, October 5, 6, 1979, at the Howard Johnson Motor Lodge, Sioux Falls, South Dakota. The following are major items of business transacted at this meeting.

1. NEW COUNCILORS AND ALTERNATES SEATED. New Councilors seated were Dr. A. A. Lampert, Jr., Brookings-Madison District, and Dr. Eldon Bell, Whetstone Valley District. Alternate Councilors seated included Dr. Curtis Wait and Dr. Joseph Kass.

2. PLACEMENT OF PHYSICIAN ASSISTANTS. The Council reaffirmed the SDSMA position that physician assistants should practice within the confines of the law and regulations as now exists under the South Dakota State Board of Medical and Osteopathic Examiners and with close physician supervision.

3. ADVANCED LIFE SUPPORT EMERGENCY MEDICAL SERVICES SYSTEM PROPOSAL FOR NORTHEAST S.D. The Council rejected the recommendation of the Commission on Medical Service to accept this proposal and encourage early implementation.

4. CONTINUING MEDICAL EDUCATION. The Council directed that an amendment to the Bylaws be submitted to the House of Delegates in June which would rescind continuing medical education as a requirement for membership in the SDSMA. However, the Council recommends that members obtain 50 hours of Category I credits every three years, and recommends to the Board of Medical and Osteopathic Examiners that continuing medical education not be required by law to maintain a license to practice medicine in South Dakota.

5. LICENSURE OF RADIOLOGIC TECHNOLOGISTS. The Council voted that the SDSMA not endorse legislation to license radiologic technologists; that the question of radiologic equipment exposure and health hazards is the responsibility of the Health Department and the diagnostic quality of radiologic films is the responsibility of the physician; and the SDSMA encourages employers to maintain high quality equipment and technicians.

6. CONTINUING EDUCATION FOR ALLIED HEALTH PROFESSIONALS. The Council favors continuing education for allied health professionals; however, the SDSMA strongly opposes any mandatory requirements for continuing education for licensure.

7. THIRD PARTY REIMBURSEMENT TO PARAMEDICALS. The Council voted to oppose direct third party reimbursement to paramedicals.

8. STUDENT MEMBERSHIP DUES AND VOTING PRIVILEGES. The Council directed that an amendment to the Bylaws be submitted to the House of Delegates in June which would require \$10 annual dues for students and would allow voting privileges for students as follows: one delegate from each class for the House of Delegates, one councilor and one representative on each of the standing commissions.

9. MEMBERSHIP DIRECTORY. The Council directed that a directory of SDSMA members be published, such directory to include the physician's name, address, specialty, telephone number, spouse's name and home address and telephone number. Only members who have submitted their dues by March 30 will be included in the directory.

10. WESTERN PHYSICIANS PURCHASING ASSOCIATION. The Council voted to withdraw endorsement of the Western Physicians Purchasing Association.

11. PROFESSIONAL LIABILITY. The Council directed that the SDSMA develop jointly with the insurance carrier, legal counsel and the Hospital Association an educational program on professional liability which can be presented at district societies.

12. JOINT PRACTICE COMMISSION. The Council directed that the SDSMA withdraw from the Joint Practice Commission, and meet with nurses and other allied health professionals to discuss matters of mutual concern as deemed necessary by the Medical Association.

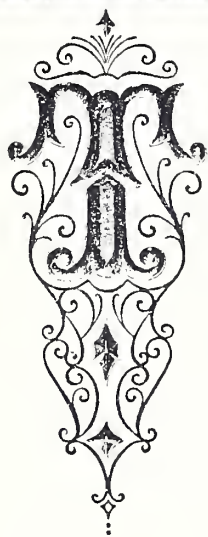
13. AMA PRINCIPLES OF MEDICAL ETHICS. The Council requested that the individual district societies review and discuss the AMA Principles of Medical Ethics along with proposed changes, that this then be discussed at an upcoming Council meeting and the AMA delegate instructed at that time regarding the recommendations of the SDSMA.

ACTION vs. INACTION

UNITE WITH

SODAPAC

If your practice is Incorporated, SoDaPAC and AMPAC voluntary political contributions should be written on a PERSONAL CHECK. Contributions are not limited to the suggested amount. Neither the AMA nor the SDSMA will favor or disadvantage anyone based upon the amounts of or failure to make PAC contributions. Contributions are subject to the limitations of FEC Regulations, Sections 110.1, 110.2 and 110.5 (Federal regulations require this notice). Copies of SoDaPAC and AMPAC reports are filed with the Federal Election Commission and are available for purchase from the Federal Election Commission, Washington, D.C.



here is no time more
appropriate than this
to say Thank You, and
to wish you a Happy
Holiday Season —

*The Staff of
South Dakota Blue Shield*



**INDEX TO VOLUME XXXII
BY TITLE, SUBJECT AND AUTHOR**

A						Month	Page
Adams, Curtis, M.D.				Proteinuria, and Abnormal Serum Proteins	Milton C. Zadina, M.D.	Nov.	25
Current Progress in Obstetrics and Gynecology				Fifteen Year Old Male and Six Month Old Male	With Marked Hypoproteinemia and Edema	Kurt E. Johnson, M.D.	
Lecture #12—Review of Breech Presentation	Feb.	15		John D. Barker, M.D.		Dec.	21
Adrian, Barton, M.D.				Barlow, John F., M.D.			
Manic-Depressive Illness or Paranoid Schizophrenia?				Two Unusual Scrotal Masses in Young Boys	John H. Hoskins, M.D.	July	7
H. T. Herman, M.D.	Feb.	7		Bean, David W., M.D.			
Aspaas, Paul K., Jr., M.D.				Widows and Widowhood	Cecilia Roberts, Ph.D.	April	9
Clinicopathological Conference				Brady, F. S., M.D.			
Twenty-Six Year Old Postpartum				Clinicopathological Conference			
Female With Thyrotoxicosis				Forty-Eight Year Old Man With Edema of the	Lower Extremities and Hodgkin's Disease		
Thomas Grau, M.D.				John F. Barlow, M.D.			
Fred Lovrien, M.D.				R. D. Willix, M.D.		Feb.	25
John F. Barlow, M.D.	June	23		Burgher, Louis W., M.D.			
Auxiliary News	May	37		Poor Correlation of Symptoms, Roentgenograms	and Pulmonary Functions in a Case of		
				Endobronchial Sarcoidosis	Jay Bachmayer, M.D.	Jan.	35
B				C			
Bachmayer, Jay, M.D.				Clinicopathological Conference			
Poor Correlation of Symptoms, Roentgenograms				Twelve Year Old Caucasian Male With	Asymptomatic Hypertension		
and Pulmonary Functions in a Case of				John F. Barlow, M.D.			
Endobronchial Sarcoidosis				James R. Reynolds, M.D.			
Louis W. Burgher, M.D.	Jan.	35		Michael Stassen, M.D.		Jan.	7
Barker, John D., M.D.				Forty-Eight Year Old Man With Edema of the	Lower Extremities and Hodgkin's Disease		
Clinicopathological Conference				John F. Barlow, M.D.			
Fifteen Year Old Male and Six Month Old Male				F. S. Brady, M.D.			
With Marked Hypoproteinemia and Edema				R. D. Willix, M.D.		Feb.	25
Kurt E. Johnson, M.D.				Thirty-Seven Year Old Man With History of Chronic	Ulcerative Colitis and Recent Onset of Jaundice		
John F. Barlow, M.D.	Dec.	21		Robert Seidel, M.D.			
Barlow, John F., M.D.				James R. Reynolds, M.D.			
Clinicopathological Conference				John F. Barlow, M.D.		March	21
Twelve Year Old Caucasian Male With				Thirty-Five Year Old Man With Swelling	of the Right Testis		
Asymptomatic Hypertension				John F. Barlow, M.D.			
James R. Reynolds, M.D.				John Hoskins, M.D.			
Michael Stassen, M.D.	Jan.	7		Dennis Foster, M.D.		April	33
Forty-Eight Year Old Man With Edema of the				Seventy-Three Year Old Woman With Recurrent	Cystitis and Pneumaturia		
Lower Extremities and Hodgkin's Disease				Roger Olsen, M.D.			
F. S. Brady, M.D.				John F. Barlow, M.D.		May	9
R. D. Willix, M.D.	Feb.	25		Twenty-Six Year Old Postpartum Female With	Thyrotoxicosis		
Thirty-Seven Year Old Man With History				Thomas Grau, M.D.			
of Chronic Ulcerative Colitis and Recent				Paul K. Aspaas, Jr., M.D.			
Onset of Jaundice				Fred Lovrien, M.D.			
Robert Seidel, M.D.				John F. Barlow, M.D.		June	23
James Reynolds, M.D.	March	21		Two Year Old Child With Pain on Urination			
Thirty-Five Year Old Man With Swelling of the				J. H. Hoskin, M.D.			
Right Testis				L. G. Thatcher, M.D.			
Dennis Foster, M.D.				D. G. Nordstrom, M.D.		Sept.	27
John Hoskins, M.D.	April	33		Seven Month Old Child With Fever, Lethargy	and Bulging Fontanel		
Seventy-Three Year Old Women With Recurrent				Thomas Huber, M.D.		Oct.	19
Cystitis and Pneumaturia				Fifty-Two Year Old Man With Urethral Bleeding,			
Roger Olsen, M.D.	May	9					
Twenty-Six Year Old Postpartum Female With							
Thyrotoxicosis							
Thomas Grau, M.D.							
Paul K. Aspaas, Jr., M.D.							
Fred Lovrien, M.D.	June	23					
Two Year Old Child With Pain on Urination							
J. H. Hoskins, M.D.							
L. G. Thatcher, M.D.							
D. G. Nordstrom, M.D.	Sept.	27					
Seven Month Old Child With Fever, Lethargy							
and Bulging Fontanel							
Thomas Huber, M.D.	Oct.	19					
Fifty-Two Year Old Man With Urethral Bleeding,							

	Month	Page
Fifty-Two Year Old Man With Urethral Bleeding, Proteinuria, and Abnormal Serum Proteins Milton C. Zadina, M.D.		
John F. Barlow, M.D.	Nov.	25
Fifteen Year Old Male and Six Month Old Male With Marked Hypoproteinemia and Edema Kurt E. Johnson, M.D.		
John D. Barker, M.D.		
John F. Barlow, M.D.	Dec.	21
Community Service Award	Aug.	41
Council Meeting Highlights March—p. 31, June—p. 37, Aug.—p. 9, Dec.—p. 29		
Coverage of a Full Thickness Heel Wound By a Muscle Transposition Flap William Lineaweaver, M.D.		
Joyce Schultz, M.D.	June	7
Current Progress in Obstetrics and Gynecology Lecture #11—Preeclampsia Loren P. Peterson, M.D.	Jan.	21
Lecture #12—Review of Breech Presentation Curtis Adams, M.D.	Feb.	15

D

Didolkar, Shailaja M., M.D. Major/Multiple Congenital Anomalies and Intrapartum Fetal Heart Rate Patterns Milton Mutch, M.D.	Sept.	5
Distinguished Service Award "Doctor Jeckel and Doctor Greed" Winston B. Odland, M.D.	Aug.	41
	March	35

E

Editorial Kennedy's New Health Proposal: More Slogan Than Reality South Dakota Medical School Endowment Association Needs You G. E. Tracy, M.D.	Aug.	44
	Nov.	21

F

Fifty Year Club Members Foster, Dennis, M.D.	Aug.	41
Clinicopathological Conference Thirty-Five Year Old Man With Swelling of the Right Testis John F. Barlow, M.D.		
John Hoskins, M.D.	April	33
Friefeld, Saul, M.D. Intramural Hematoma of the Duodenum, A Case Report and Literature Review Bruce C. Lushbough, M.D.		
Joseph Primrose, M.D.	May	31
Future Meetings Jan.—p. 41, Feb.—p. 37, March—p. 41, April—p. 43, May—p. 39, June—p. 40, July—p. 32, Aug.—p. 54, Sept.—p. 34, Oct.—p. 42, Nov.—p. 38, Dec.—p. 36		

G

Grau, Thomas, M.D. Clinicopathological Conference Twenty-Six Year Old Postpartum Female With Thyrotoxicosis Paul K. Aspaas, Jr., M.D.		
Fred Lovrien, M.D.		
John F. Barlow, M.D.	June	23

H

Harris, Russell, M.D. President's Page April—p. 21, May—p. 19		
---	--	--

Month Page

Hermann, H. T., M.D. Manic-Depressive Illness or Paranoid Schizophrenia? Barton Adrian, M.D.	Feb.	7
Hoskins, John, M.D. Clinicopathological Conference Thirty-Five Year Old Man With Swelling of the Right Testis John F. Barlow, M.D.		
Dennis Foster, M.D.	April	33
Two Year Old Child With Pain On Urination L. G. Thatcher, M.D.		
D. G. Nordstrom, M.D.		
John F. Barlow, M.D.	Sept.	27
Hoskins, John, M.D. Two Unusual Scrotal Masses in Young Boys John F. Barlow, M.D.	July	7
Huber, Thomas, M.D. Clinicopathological Conference Seven Month Old Child With Fever, Lethargy and Bulging Fontanel John F. Barlow, M.D.	Oct.	19

I

Intramural Hematoma of the Duodenum, A Case Report and Literature Review Bruce C. Lushbough, M.D.		
Saul Friefeld, M.D.		
Joseph Primrose, M.D.	May	31

J

Johnson, Kurt E., M.D. Clinicopathological Conference Fifteen Year Old Male and Six Month Old Male With Marked Hypoproteinemia and Edema John D. Barker, M.D.		
John F. Barlow, M.D.	Dec.	21

K

Kaatz, Brian L., Pharm. D. Review of Sympathomimetic Bronchodilators	March	9
Kennedy's New Health Proposal: More Slogan Than Reality Editorial	Aug.	44

L

Laboratory Aids South Dakota Pathology Society Page Jan.—p. 18, Feb.—p. 10, March—p. 32, April—p. 16, May—p. 30, June—p. 15, July—p. 24, Aug.—p. 37, Sept.—p. 17, Oct.—p. 40, Nov.—p. 32, Dec.—p. 18		
Letters to the Editor Jan.—p. 32, May—p. 20, June—p. 34, Nov.—p. 12		
Lineaweaver, Wm., M.D. Coverage of a Full Thickness Heel Wound by a Muscle Transposition Flap Joyce Schultz, M.D.	June	7
Lovrien, Fred, M.D. Clinicopathological Conference Twenty-Six Year Old Postpartum Female with Thyrotoxicosis Thomas Grau, M.D.		
Paul K. Aspaas, Jr., M.D.		
John F. Barlow, M.D.	June	23
Lushbough, Bruce C., M.D. Intramural Hematoma of the Duodenum, A Case Report and Literature Review Saul Friefeld, M.D.		
Joseph Primrose, M.D.	May	31

	Month	Page
M		
Major/Multiple Congenital Anomalies and Intrapartum Fetal Heart Rate Patterns Shailaja Didolkar, M.D.		
Milton Mutch, M.D.	Sept.	5
Manic-Depressive Illness or Paranoid Schizophrenia? Barton Adrian, M.D.		
H. T. Herman, M.D.	Feb.	7
Mutch, Milton, M.D.		
Major/Multiple Congenital Anomalies and Intrapartum Fetal Heart Rate Patterns Shailaja M. Didolkar, M.D.	Sept.	5
Minutes of the House of Delegates Aug.—p. 10		

N		
Nordstrom, D. G., M.D.		
Clinicopathological Conference Two Year Old Child with Pain on Urination J. H. Hoskins, M.D.		
L. G. Thatcher, M.D.		
John F. Barlow, M.D.	Sept.	27

O		
Odland, Winston B., M.D.		
"Doctor Jeckel and Doctor Greed"	March	35
Olsen, Roger, M.D.		
Clinicopathological Conference Seventy-Three Year Old Woman with Recurrent Cystitis and Pneumaturia John F. Barlow, M.D.	May	9

P		
Peik, Donald J., M.D.		
X-Ray Case of the Month Peteireit, Martin F., M.D.		
April—p. 24, July—p. 14, Oct.—p. 30		
Peteireit, Martin F., M.D.		
X-Ray Case of the Month Peik, Donald J., M.D.		
April—p. 24, July—p. 14, Oct.—p. 30		
Petersen, Loren P., M.D.		
Current Progress in Obstetrics and Gynecology Lecture #11—Preeclampsia	Jan.	21
Physician and the Hyperactive Child, The Carl N. Rutt, M.D.	Nov.	7
Poor Correlation of Symptoms, Roentgenograms and Pulmonary Functions in a Case of Endobronchial Sarcoidosis Jay Bachmayer, M.D.		
Louis W. Burgher, M.D.	Jan.	35
President's Page Russell Harris, M.D.		
April—p. 21, May—p. 19		
Duane Reaney, M.D.		
June—p. 10, July—p. 21, August—p. 27, Sept.—p. 15, Oct.—p. 29, Nov.—p. 19, Dec.—p. 15		
Primrose, Joseph, M.D.		
Intramural Hematoma of the Duodenum, A Case Report and Literature Review Saul Friefeld, M.D.		
Bruce C. Lushbough, M.D.	May	31

R		
Ranney, Brooks, M.D.		
Study of Endometrial Carcinoma and Potential Precursor Lesions, A (Part 1)	Dec.	5

S		
Reaney, Duane, M.D.		
President's Page June—p. 10, July—p. 21, Aug.—p. 27, Sept.—p. 15, Oct.—p. 29, Nov.—p. 19, Dec.—p. 15		
Reimplantation of Nearly Amputated Upper Extremity: Long-term Followup Robert E. Van Demark, M.D.	Oct.	11
Review of Sympathomimetic Bronchodilators Brian L. Kaatz, Pharm. D.	March	9
Reynolds, James R., M.D.		
Clinicopathological Conference Twelve Year Old Caucasian Male with Asymptomatic Hypertension John F. Barlow, M.D.		
Michael Stassen, M.D.	Jan.	7
Reynolds, James R., M.D.		
Clinicopathological Conference Thirty-Seven Year Old Man with History of Chronic Ulcerative Colitis and Recent Onset of Jaundice Robert Seidel, M.D.		
John F. Barlow, M.D.	March	21
Roberts, Cecilia, Ph.D.		
Widows and Widowhood David Bean, M.D.	April	9
Roster—Alphabetical	Aug.	50
Roster—By District	Aug.	47
Rutt, Carl N., M.D.		
Physician and the Hyperactive Child, The	Nov.	7
S		
Schultz, Joyce, M.D.		
Coverage of a Full Thickness Heel Wound By a Muscle Transposition Flap Wm. Lineaweaver, M.D.	June	7
Seidel, Robert, M.D.		
Clinicopathological Conference Thirty-Seven Year Old Man with History of Chronic Ulcerative Colitis and Recent Onset of Jaundice James R. Reynolds, M.D.		
John F. Barlow, M.D.	March	21
South Dakota Academy of Family Physicians Chapter News Jan.—p. 29, Feb.—p. 21, March—p. 15, April—p. 18, May—p. 23, June—p. 17, July—p. 23, Aug.—p. 29, Sept.—p. 17, Oct.—p. 33, Nov.—p. 37, Dec.—p. 17		
South Dakota Medical School Endowment Association Needs You		
Editorial G. E. Tracy, M.D.	Nov.	21
South Dakota Pathology Society Page Laboratory Aids Jan.—p. 18, Feb.—p. 10, March—p. 32, April—p. 16, May—p. 30, June—p. 15, July—p. 24, Aug.—p. 37, Sept.—p. 22, Oct.—p. 40, Nov.—p. 32, Dec.—p. 18		
Stassen, Michael, M.D.		
Clinicopathological Conference Twelve Year Old Caucasian Male with Asymptomatic Hypertension John F. Barlow, M.D.		
James R. Reynolds, M.D.	Jan.	7
Study of Endometrial Carcinoma and Potential Precursor Lesions, A (Part 1) Brooks Ranney, M.D.	Dec.	5

T

Thatcher, L. G., M.D. Clinicopathological Conference Two Year Old Child with Pain on Urination J. H. Hoskins, M.D. D. G. Nordstrom, M.D. John F. Barlow, M.D.	Sept.	27
This Is Your Medical Association Jan.—p. 31, Feb.—p. 18, April—p. 23, June—p. 21, Aug.—p. 43, Oct.—p. 35		
Tracy, G. E., M.D. South Dakota Medical School Endowment Association Needs You Editorial	Nov.	21
Transactions of the South Dakota State Medical Association Ninety-Eighth Annual Meeting	Aug.	7
Two Unusual Scrotal Masses in Young Boys John H. Hoskins, M.D. John F. Barlow, M.D.	July	7

V

Van Demark, Robert E., M.D. Reimplantation of Nearly Amputated Upper Extremity: Long-term Followup	Oct.	11
--	------	----

W

Widows and Widowhood David W. Bean, M.D. Cecilia Roberts, Ph.D.	April	9
Willix, R. D., M.D. Clinicopathological Conference Forty-Eight Year Old Man with Edema of the Lower Extremities and Hodgkin's Disease John F. Barlow, M.D. R. S. Brady, M.D.	Feb.	25

X

X-Ray Case of the Month Donald J. Peik, M.D. Martin F. Peterreit, M.D. April—p. 24, July—p. 14, Oct.—p. 30		
---	--	--

Z

Zadina, Milton C., M.D. Clinicopathological Conference Fifty-Two Year Old Man with Urethral Bleeding, Proteinuria, and Abnormal Serum Proteins John F. Barlow, M.D.	Nov.	25
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Sioux River Valley Community Health Center

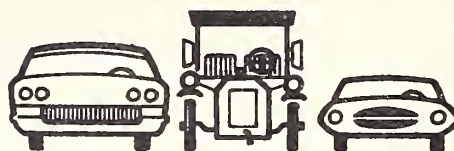
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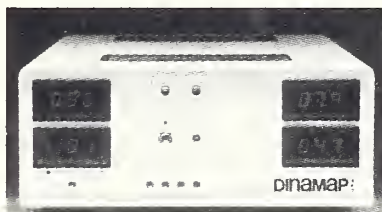
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SD

Future Meetings

January

American Society of Clinical Pathologists Joint Meeting with College of American Pathologists, San Diego, CA, Jan. 4-11. Contact: The Amer. Soc. of Clinical Pathologists, 2100 W. Harrison St., Chicago, IL 60612. Phone: (312) 738-1336.

Clinical Pharmacology for the Practitioner, Maui Intercontinental Hotel, Maui, HI, Jan. 6-11. 21 hrs. AMA Category 1 and AAFP credits. Contact: Amer. Inst. of Postgraduate Ed., Box 21488, Seattle, WA 98111. Phone: (206) 392-7939.

AMA Winter Scientific Meeting, San Antonio, TX, Jan. 12-15. Contact: AMA, 535 N. Dearborn St., Chicago, IL 60610.

Pediatrics in Review — 1980 — The School Age Child, Acapulco, Mex., Jan. 13-19. Contact: Judson Hawk, Jr., M.D., Scottish Rite Hosp. for Crippled Children, 1001 Johnson Ferry Rd., Atlanta, GA 30342. Phone: (404) 256-5252.

Mayo Foundation Outreach Seminar, McKennan Hosp. Aud., Sioux Falls, SD, Jan. 18-19. AMA Category 1 and AAFP credits. Contact: Off. of Med. Ed., McKennan Hosp., 800 E. 21st St., Sioux Falls, SD 57101.

American College of Allergists, Americana Hotel, Bal Harbour, Miami Beach, FL, Jan. 19-23. AMA Category 1 credits. Contact: Frances P. White, Exec. Sec., Am. College of Allergists, 2141-14th St., Boulder, CO 80302. Phone: (303) 447-8111.

Heredity Acquired Neurological Disorders, Coffman Union Theater, U. of Minn., Minneapolis, MN, Jan. 24-25. Fee: \$100. 13 hrs. AMA Category 1 credits. Contact: Cont. Med. Ed., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Coronary Disease, Exercise Testing and Cardiac Rehabilitation, Konover Hotel, Miami, FL, Jan. 25-27. 13 hrs. AMA Category 1 credits. Contact: IMEC, 64 Inverness Dr., E., Englewood, CO 80112. Phone: 800-525-8646, Ext. 236.

EKG Interpretation & Arrhythmia Management, Desert Inn, Las Vegas, NV, Jan. 25-27. 15 hrs. AMA Category 1 credits. Contact: IMEC, 64 Inverness Dr., E., Englewood, CO 80112. Phone: 800-525-8646, Ext. 236.

Cardiac Rehabilitation, Le Pavillion, New Orleans, LA, Jan. 25-27. 13 hrs. AMA Category 1 credits. Contact: IMEC, 64 Inverness Dr., E., Englewood, CO 80112. Phone: 800-525-8646, Ext. 236.

Vail Midwinter Cancer Seminar, Vail, CO, Jan. 30-Feb. 1. 10 hrs. AMA & AAFP Category 1 credits. Contact: Midwinter Cancer Seminar, Amer. Cancer Society, 1809 E. 18th Ave., Denver, CO 80218. Phone: (303) 321-2464.

February

Cardiac Ischemia and Arrhythmias — Current Concepts for Diagnosis and Treatment, Bahai Resort, San Diego, CA, Feb. 1-3. 13 hrs. AMA Category 1 credits. Contact: IMEC, 64 Inverness Dr., E., Englewood, CO 80112. Phone: 800-525-8646, Ext. 236.

Midwinter Family Practice Update, Boyne Highlands Ski Resort, Harbor Springs, MI, Feb. 3-8. Contact: Off. of Cont. Med. Ed., Towsley Center for Cont. Med. Ed., U. of Mich. Med. School, Ann Arbor, MI 48109. Phone: (313) 764-2287.

Coronary Disease, Exercise Testing and Cardiac Rehabilitation, Mountain Shadows Resort, Scottsdale, AZ, Feb. 8-10. 13 hrs. AMA Category 1 credits. Contact: IMEC, 64 Inverness Dr., E., Englewood, CO 80112. Phone: 800-525-8646, Ext. 236.

Advanced Problems in Cardiac Emergencies, Snowbird Resort, Utah, Feb. 9-14. 21 hrs. AMA & AAFP Category 1 credits. Contact: American Institute of Postgraduate Ed., Box 21488, Seattle, WA 98111. Phone: (206) 392-7939.

Third Annual Vail Urology Conference, Kiandra-Talisman Lodge, Vail, CO, Feb. 9-16. Fee: \$240. 22 hrs. AMA Category 1 credits. Contact: Third Annual Vail Urology Conf., Box 11366, Denver, CO 80211. Phone: (800) 525-5810.

Second Annual Vail Emergency Medicine/Critical Care Conference, Lion Square Lodge, Vail, CO, Feb. 9-16. Fee: \$240. 22 hrs. AMA

Category 1 credits. Contact: Second Annual Vail Emer. Med./Critical Care Conf., Box 11366, Denver, CO 80211. Phone: (800) 525-5810.

Epidemiology for the Occupational Physician, Gjenelefe Resort, Cypress Gardens, FL, Feb. 12-16. Fee: \$265. 24 AMA Category 1 credits. Contact: Don Hoops, Ph.D., Dir. of Educ., Amer. Occupational Med. Asso., 150 N. Wacker Dr., Chicago, IL 60606. Phone: (312) 782-2166.

Sixth Annual Vail OB/GYN Conference, The Mark, Vail, CO, Feb. 16-23. Fee: \$240. 22 hrs. AMA Category 1 credits. Contact: Sixth Annual Vail OB/GYN Conf., Box 11366, Denver, CO 80211. Phone: (800) 525-5810.

Fifth Annual Vail Psychiatry Conference, Lion Square Lodge, Vail, CO, Feb. 16-23. Fee: \$240. 22 hrs. AMA Category 1 credits. Contact: Fifth Annual Vail Psy. Conf., Box 11366, Denver, CO 80211. Phone: (800) 525-5810.

First Annual Vail Pathology Conference, Lion Square Lodge, Vail, CO, Feb. 16-23. Fee: \$240. 22 hrs. AMA Category 1 credits. Contact: First Annual Vail Path. Conf., Box 11366, Denver, CO 80211. Phone: (800) 525-5810.

Family Practice Review: Update 1980, Radisson St. Paul Hotel, St. Paul, MN, Feb. 18-22. 45 hrs. AMA Category 1 credits. Contact: Cont. Med. Ed., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Coronary Disease, Exercise Testing and Cardiac Rehabilitation, Sahara Hotel, Las Vegas, NV, Feb. 22-24. 13 hrs. AMA Category 1 credits. Contact: IMEC, 64 Inverness Dr., E., Englewood, CO 80112. Phone: 800-525-8646, Ext. 236.

Cardiology Today, U. of Iowa Hosp., Iowa City, IA, Feb. 25-28. Fee: \$275. 30 hrs. AMA Category 1 & AAFP credits. Contact: Carl White, M.D., Cardiovascular Div., U. of Iowa Hosp., Iowa City, IA 52242. Phone: (319) 356-3413.

March

Mediclinics Spring Seminars, Fort Lauderdale, FL., March 3-14. Fee: \$325. 50 hrs. AMA & AAFP Category 1 credits. Contact: Mediclinics, 832 Central Medical Bldg., St. Paul, 55104.

